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The Halford Oration.[1]

"BEFORE AND AFTER."

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Professor of Physiology and Dean of the Faculty of Medicine in the University of Melbourne.

LIKE many things in these times, the title of my address is now behind the times. I was asked for the title early this year and there was only one subject which seemed worth while—the development of our School of Medicine. Such a subject is worth while at any time, but it was of pressing importance to me because I had just succeeded to the two offices first held in this school by the man whom we honour tonight—George Britton Halford, first professor of physiology and dean of the Faculty of Medicine in this University. For many years in the School of Medicine here, there has been a feeling that all was not best with us. I have been involved in the deliberations of many of the committees which have considered this matter over the years. Early this year I felt that our efforts were best summed up by the words of Shelley in his *Ode to a Skylark*: "We look before and after, and pine for what is not." Many of you will recognize there the activity of a committee. Forwards and backwards the verbal battle surges as the war goes on against a non-existent enemy; the gallant gaze to the right, the surreptitious glance to the left, the tumult and confusion in the swirling cloud of words until at last decision is reached—to refer it to a subcommittee. That is when one can only pine for what is not; executive action appears to be impossible.

Since I gave the title of my address something has happened. The forces for the advancement of medicine in the University have agreed upon the main objective and the route to be followed in achieving it. There is now little time to pine; there is coherence and comradeship in our school. But lest we become too optimistic, let us consider our history with the hope that we can learn its lessons.

In 1850, Victoria, with a population of 75,000 people, was declared a colony separate from New South Wales. It had a unicameral legislature called the Legislative Council. Shortly thereafter gold in quantity was discovered in the colony, and the population and ambitions of the colony rose on the tide of apparent wealth. To most it represented an opportunity to indulge the lower appetites; to some it gave the opportunity to set up large houses in the style of the day and to fill them with red plush furniture. To others it presented the opportunity to found a university. This cause was taken up by the Attorney-General, Mr. H. C. E. Childers, the Lieutenant-Governor, Mr. C. J. LaTrobe, and Mr. William Stawell. On January 7, 1853, the Bill to incorporate and endow the University of Melbourne passed its second reading in the legislature. Note the date: on January 7 a bill passed its second reading—nowadays, if the Prime Minister on January 7 wanted to declare war he would be hard pressed to get a quorum! At that time the population of Victoria had more than trebled in three years and was to increase rapidly over the decade. No doubt there were problems concerned with housing, sanitation and supplies. Prob-

[1] The sixteenth Halford Oration, delivered at the University of Melbourne on August 7, 1950.

ably people returned to England and wrote letters to papers complaining that the gold was not delivered in the penny post to disembarking migrants! But I dwell on this period of our inauguration for a moment to let you contemplate the phenomenon of human endurance. For greed, men and women of all estates overcame hardship and danger in their headlong drive. As a feat of human endeavour and endurance in this country it has been equalled only in time of war. It has never been approached in the peaceful development of this country.

In this atmosphere of "Sydney or the bush" the courses of the University began in a temporary building. (It must have been a rabbit hutch—its progeny are now all over the campus!) The *Morning Herald*, with that devotion to higher learning which sometimes attaches to a daily paper, went on record with the statement that "a University ought to be the nursery of great men, of those on whom, whatever be our form of government, our true permanent welfare depends", which appears to attach undue importance to our function in training civil servants!

In 1855 Dr. Anthony Colling Brownless became a member of Council. He exerted himself for the foundation of a medical school and was asked to prepare details. These he quickly provided, with an estimate for buildings and equipment at £21,000. This was received sympathetically by the Chief Secretary, but turned down by the Government because the time was not ripe. Four years later the project was again put forward with the same result. In the following year Council again urged the plan, but reduced the request to £12,000. Committees met and agreed that the time was ripe, but that £4000 would be sufficient. The Government now indicated that they agreed the time was ripe, but that revenues were dangerously low and the money could not be found. The Council, with great courage, however, and acting on an unofficial assurance of support, proceeded to legislation for the school of medicine, and by imposing on private citizens and filching from the salaries of other members of staff, provided instruction in the first year of the course. Further, they appointed George Britton Halford, M.D., M.R.C.S., to be professor of anatomy, physiology and pathology, and even sent to him £500 to buy equipment for his school. Halford was well and favourably known for his researches in physiology and came with the recommendations of James Paget, Richard Owen and the learned journals of England. One must conclude that he was a man of courage—he came to a school with no buildings and no assured income, no old-age pension and no medical benefits. His building plans were hacked and compressed and even in that form did not secure the immediate support of the Government. As a result, the first classes in anatomy and dissection were begun in a shed in the yard of the professor's residence. In paying tribute to George Britton Halford we must pay tribute to his wife, for in 1863, when the shed was in the yard, antiseptics were practically unknown. Pasteur and Lister had not appeared over the medical horizon.

Later in the year 1863 an amount of £5700 was provided by the Government for the buildings, which had been reduced to the bare minimum. The failure to provide for imminent expansion was later costly and produced appalling results æsthetically, but we have done worse since then. The building was completed in May of the next year, and the courses have been held there ever since. One can still see the original floors and trace out the various rooms in my department. The study which I took over seventy-five years later still has Halford's table in it. Cut into its surface are the three holes in which his microscope stood securely. These holes often conjure up in my mind the first introduction to the colony of Victoria of what is still a standard instrument of science. I imagine that the eager people of the last century came to the microscope with their specimens, for Halford knew only too well what would be the fate of his instrument if he let it leave his study in confident but unskilled hands.

Enough of musing. A school had been born. Every excuse except one had been used to prevent it—the time is not ripe, funds are low—familiar to everyone who seeks to advance learning. The excuse which was not used was: "The man is not available". The medical profession, working in ardent collaboration with the Chancellor, found a man of courage and vision. Under his guidance the school not only was born but survived, and has now become a living influence in the field of medicine. Some would bemoan the fact that when the early struggles were over dissension and conflict arose on a number of occasions between the hospitals and the University. It will be a sorry day when disinterested disputation ceases in the medical school.

Our medical school is a school of a university. So long as it is in the University all men who have the interests of learning at heart must uphold the ideal of the University. I make so bold as to define this ideal as the recognition and teaching of what is held to be true and the enlargement of this field. Our aim must therefore be to seek the facts and to join them in the simplest theoretical synthesis which can be developed. We hold it as our duty to impart with all honesty our results to our colleagues and students, so that they, standing on our shoulders, may reach higher and further into the currents of human understanding of the universe, even to human understanding of man himself. If we adopt this definition as the basis for work in a university we do not need to argue those spurious alternatives of teaching and research—at the university level teaching can be contemporary and alive only when the teacher is a searcher; every research worker teaches even if only by writing.

From this consideration of our creed two matters of importance arise at the present time: the question of secrecy and the question of our future in the University. We have stated that we should impart the knowledge which we are helping to develop. This is part of the Hippocratic oath—remember it?

> I will impart a knowledge of the Art to my own sons and those of my teachers and to those bound by a stipulation and oath according to the law of medicine, but to none others. I will follow that system of regimen which, according to my ability and judgement, I consider for the benefit of my patients and abstain from whatever is deleterious and mischievous.

At the present time there are forces acting toward reticence about medical knowledge. The one branch of learning which has always been international should remain so. If the British and American Medical Associations invited the Russian counterpart to collaborate in organizing a world medical congress, who knows what good might come? It would be a safe overture—no harm could result. I know that everybody hates war, but there is also the stage when subgovernmental organizations will not move for fear of popular displeasure. Please do not mistake my meaning. If the practitioners of a country are not bound by the laws of medicine, they are not of our brotherhood and get information as they may; if they are of our brotherhood there will be no secrets. You may ask, however, what of the secrets of those doctors who design death. When they do so they are not of the brotherhood of medicine. That does not mean that they are not serving their nation well. But in such pursuits they pass out from our brotherhood, and the laws of the brotherhood neither protect them nor condemn them in their own choice.

We now come to consider whether medicine should continue in a university or move to the periphery as a technological pursuit. Medicine preceded all other branches of learning in developing organized teaching and investigation. It preceded them also in developing general hypotheses upon which to base practice and extend knowledge. Rarely did it elevate these hypotheses to a place of reverence, and as a consequence it has at times enjoyed slighting references to our inexactness by the younger groups such as physicists and chemists. Now they are going through their first real confusion and will realize that medical laws are just as important as physical laws, but we today have been too long at the game to fall into the trap of dogma. Apart from the contribution of medicine to the subject matter of the intellectual, we must also consider it from the point of view of the interest it has for the other branches of learning—both in physics and chemistry there is a rediscovery of the compelling interest of medical biology, which gave rise to those

sciences in the first place. The health problems of the community and the vast store of sociological knowledge which is the birthright of medicine make it an invigorating partner in any university. We will not profit medicine or the other branches of learning by breaking our long association. Helplessness in the presence of disease is a great spur and an almost certain preventive of the sterility of scholasticism.

A subject subsidiary to this association of medicine with other branches of learning in a free institution is the question of full-time research as opposed to combined research and teaching. I have referred to it briefly in another place, but what I am about to say refers particularly to an Australian problem. Non-teaching institutions in which graduates of universities do research to the best of their ability are supported by governments by an amount of money almost double that given to universities. As a consequence university staffs are weighed down by the teaching duty. As a further consequence a great section of the scientific population of Australia is shut off from contact with the student population of the country. Both the student and the worker lose as a result. For the full development of the intellectual capacity of our students and workers we must aim at much more free association between them. This cannot happen when half the scientists of a country can be seen at work by only those young graduates who accept appointment in the closed organization in which the worker is engaged. You appreciate that I am referring to the Commonwealth Scientific and Industrial Research Organization. Their relationships to the university must be much closer than now if Australia is to get full benefit from the mature scientists in the country. You may suggest that I should stick to my last. These considerations are important in medicine, because there is a doctrine in this country that teachers should teach and research workers should do research. That doctrine should apply to the 25% of teachers who cannot do research and the 25% of research workers who cannot teach. For the others, contact with intelligent sceptical students is mutually beneficial and essential for the development of the new generation of scientists. I believe that medicine in this country will suffer if we develop the same disproportion between support for whole-time research teams and teaching-research teams as we find in other branches of science here.

For every man there are different ideal conditions for his continuing development, but there is an essential basal organization for all. The line of authority must be defined, usually some colleagueship is necessary, and buildings and supplies are essential. We have almost achieved this in Melbourne in the five departments of the medical school. In the clinical field we have not. This is the largest problem confronting the school and I shall now discuss it.

Ever since the school was founded the whole of the clinical teaching in medicine and surgery has been done by people in active practice in the city. At one stage persons elected by the University from among the honorary staffs of the teaching hospitals occupied chairs in medicine and surgery, but we have never had full-time teachers in these subjects. The results, as judged by the competence of the practice of our graduates, have been good—this is not my judgement, but the judgement of visiting medical notables. You will notice that I did not describe the results as perfect. In the practice of a profession, when life is short, the art is long and judgement is difficult, there will never be a perfect result. Too many people are inclined, until they have really practised medicine, to decry less than a 100% performance. We can justly believe that the practice of medicine by our graduates is comparable with that of the graduates of other schools. It will remain so just so long as our teaching practices are as advanced as those in other schools; it might conceivably go ahead of other schools if we developed in this school a new approach to the problem.

We have achieved standards at the level of many universities in which there are professors of medicine and surgery, precisely, I believe, because these professors are carrying out a function of training directly for practice; the mere professorial title does not equip them better for this function that the day-to-day problems of practice equip our practitioners. Indeed, I believe that no academic person can hope to approach, much less surpass, the top flight of practising doctors in the teaching of the practice of medicine and surgery. This being the case, is there then a fruitful field for the application of academic skills in the medical school? I believe there is, but I will not say in which way until I have surveyed briefly the development of medicine over the last hundred years.

A century ago some diseases were recognized, a few medicaments were effective, and surgery was restricted to incisions, amputations and a few other tests of dexterity. You will recall that general anæsthesia had just been discovered. Pasteur was developing the notion of infection and immunity. The line of development from his discoveries led to Lister; antiseptic and later aseptic surgery were the result. To the lay person the dramatic situations of surgery suggest modern surgery as the greatest product of Pasteur's discoveries. But the really great results are in the prevention of infectious disease, the cure of many by medicament and our greatly increased knowledge of living processes. Let us trace the highlights of these developments. You will recall that Pasteur applied the principle of vaccination, known for over a century, to the prevention of infection by bacteria. He also used the system of controlling inherited infection by breeding from disease-free stock. These two principles are applied at the present time to man and animals all over the world. His discovery also made obvious the principle of breaking the chain of infection. Sanitation and the safeguarding of foodstuffs are the result. Then came the discovery of toxins and antitoxins. Knowledge of these processes is still incomplete, but by the turn of the century a few simple theoretical notions about the reactions between the infective agent and the infected object had changed the manner of life in the world. Chemistry as applied to living things had been developing for sixty years, and now came the genius of Ehrlich to provide drugs tailored to the infecting agent—a simple notion, but from it has sprung a great stream of understanding of living processes and a great success in combating disease. Domagh was using the principle when he thought of using "sulpha" drugs for treatment, and we have recently had a visit from Professor Adrien Albert, who is coming back soon to the National University and is one of the great modern explorers in this field. Florey went back to nature for his chemicals when he sought to purify the substance by which the mould penicillin destroys the disease germ Staphylococcus. You know what a plum he pulled from that pie, but that is the beginning of the story, not the end of it. Other useful antibiotics have been discovered, but perhaps more important than this, such substances can be used to unlock the secrets of living processes—already a new window is opening on to the field of protein metabolism.

Those, then, are the ideas which have come in direct line from Pasteur's positive observations. They sound a very flimsy structure upon which thousands toil and millions of lives depend. Man's power to help his fellows grows in proportion to his understanding. Leonardo da Vinci expressed the relationship: "Great love is the daughter of great knowledge."

Let us take a brief glance at another field of progress. When bacteriology developed it was soon found that not all diseases were due to infection. Indeed, it should have been recognized from the work of Woodall in 1639 that there is a substance in lemon juice which is necessary to prevent scurvy and will cure it if it has developed. Many others repeated the observations, but I suspect that it was not until the germ theory started to fill in the pattern of the cause of disease that there was that confidence in inference which allowed Grijns to state the concept of a specific syndrome due to the absence of a specific substance from the diet. Eleven years later Funk stated the vitamin theory. That was less than forty years ago. You have seen scurvy, rickets, beri-beri, pernicious anæmia, pellagra and a number of other diseases come under control. The information about living processes which has developed from those studies is the basis of

understanding many phenomena other than the specific diseases, and I venture to suggest that we shall see the day when much of the performance of the central nervous system will be understood in terms of this knowledge.

I will not itemize in the other fields of great advance. The edges of theory and practice are being pushed out into the unknown by thousands of workers; the publications are many thousands of pages per month. The system of publication is not designed to portray the general pattern to the reader. It would be redundant to present argument that because a good doctor deals with a whole organism, the best doctor will be the one with the fullest comprehension of the processes of health and disease in a whole organism. Such a doctor would be the ideal product of the University if his practice was founded upon a general theoretical synthesis of medical knowledge, for he is continuously in a research situation demanding an understanding of man both as an individual and as a social being. Is such a man likely to get a general synthesis of medical knowledge from a training in a school staffed by specialists? He may do so fortuitously, but his quality would need to be exceptional. I believe that it is possible to plan so that many graduates, and not only the exceptional and lucky, can have this advantage. The knowledge of cause and process in disease is now sufficiently developed to allow a general treatment of this knowledge. Hæmorrhage sets up the same disturbances and reactions whether it occurs in a condition agreed to be medical, surgical or obstetrical. Alkalosis is the same condition no matter whether the patient is in a medical or surgical ward. Allergic manifestations present the same general features in hydatid disease or asthma. Fever is fever with its characteristic disturbances, no matter to what condition it is due. Pain is the same scourge of man and his bodily processes, whether due to the whip, the scalpel or disease. Each one of these features of disease and many more are now amenable to a synthesis of knowledge and to research. A student who had a thorough course in these general processes would be half-way to being a medical doctor. Such courses would not substitute for the sound bedside teaching which the practising specialist only can give. They would complement this training and make both the comprehension and the expansion of medical knowledge easier of achievement.

My view is, therefore, that if and when this University appoints officers in the clinical field, their duties should be the development and teaching of a theoretical synthesis in medicine. That would be a proper academic function in a field where the present stage of development of knowledge permits such treatment and indeed is crying out for it. It has practical value in that it would draw together into a sound fabric the threads collected by the student from his specialist teachers. Once the pattern of the fabric is sufficiently clear, the extension of knowledge into the unknown must proceed apace, for the known pattern will be as an outline map to an explorer in an unknown land.

I have tried to show that the time is ripe for such a development. Now all we need is the money and the men. I refuse to believe that Australia, Victoria or Melbourne is so far behind the times as to refuse funds for such a development. Capital cost would probably not exceed £500,000 and annual expenses £100,000. We readily approve hospital building schemes costing millions of pounds with running costs proportionately high. The cost of this school would be less than 5% of either the capital or running costs of the chief metropolitan hospitals. It would be the factor which makes the difference between very good and excellent doctoring in the community. It might bring some really startling advance in medicine. We cannot afford to let our men continue to stay away from the wheels of medical progress. Who will provide the money? I expect the Government will have to do so. This is not because I do not believe in private enterprise, but because I have never had the opportunity to see it tackle a large job of working for an idealistic abstraction. It might be retorted that realism is needed: I suggest that realism on its own is sufficient for those without vision or with an ax to grind. The House of Commons is just a building, but every corner and scroll harbours a human endeavour; most of them are dull, but there are many gilded by an ideal attained. These are what makes the place famous. It is easy to see the significance of a tack in the lung and to get funds for it; it is harder to see the significance of a new theory and to labour to make it possible even before it is conceived. Whether the money comes from individuals or the State or both, let us remember that a school of learning is stronger and more lasting than empires, and he who helps to create one has done the greatest good for man. Let him who will contest this contemplate the history of medicine. The school of medicine at Edinburgh has been a force in medicine ever since it was set up. It has seen Scottish independence, union, the Commonwealth, the Restoration, and the rise of democracy. It retains its essential form, for medical institutions are founded upon an abstract ideal more powerful than any written contract.

Are there the men for the job? In 1860 our forefathers looked across the world and found a man of courage and enterprise. Why should we not look for one?

I have indicated what I believe to be the major reform now necessary in the existing subject matter of the school. There are two other matters affecting more the spirit of medicine. The first one concerns the fact that medicine is practised in a human society and that medical men are a group in that society. This is a proper study for the undergraduates. Those in opposition will repeat the cliché that a doctor is concerned with medicine, not law. Any citizen is concerned with law, and it will be a sorry day if the medical profession rises above or sinks below the law. At present it might be salutary if our profession had an elementary course on the nature of law in society. We might then see our profession initiating good law instead of always being on the defensive. Ours is the basic learned profession giving the most direct service to our fellows; the organization for giving will change with the changing pattern of society. As a profession with a mean intelligence quotient of 114, we should without great labour be able to understand social organization well enough to plan our practice so that the citizens get full measure of relief from fear and disease, while we retain the freedom to advance our science and perfect our art.

Let us savour again that phrase: advance our science and perfect our art. From Egypt, Greece, Rome, Arabia, Italy, England, France, Germany, Russia, America and every country of the world, the names of men roll forth: Aristotle, Hippocrates, Galen, Rhases, Vesalius, Harvey, Pasteur, von Helmholtz, Virchow, Lister, Koch, Bernard, Pavlov, Liebig, Ehrlich. These names roll across the heavens. Yet to how many medical graduates would they mean anything? If our graduates are to appreciate the science and glory in their art they must be aware of the events of medicine in history. We could start a section of medicine in history tomorrow; all that is required is the conviction that it is worth while. The monetary outlay would be small compared with the effect upon the 10% of students whose interest would be excited.

We have indulged in self-criticism. It is said to be a healthy thing to do. I do not believe that it is if the only result is a sigh. The medical school in Melbourne is on the edge of the most fertile period of its existence. The University is at one with the clinical schools in the metropolitan hospitals in developing university departments at the clinical level. It is an exciting prospect. With the goodwill and the practical support of the community it will succeed. The quality of the students in our courses makes this certain. Halford, Allen, Berry, Osborne and MacCallum, with the loyal and wise support of many great medical men of this city, have kept our school on a true course and brought it to a level commensurate with the material and intellectual resources of the community. We who now take up its direction will realize that there is no automatic compass nor jet propulsion. Progress will come steadily from constant expenditure of energy. Every mind must give its wisest counsel, and our loyalties must be true. Our ambitions require that our discussions should be frank. The time is ripe. The community cannot afford to neglect the opportunity. We must seek the men. Let the vision and accomplishment of Halford be our inspiration!

THE INDICATIONS FOR SPLENECTOMY: A DISCUSSION OF SOME MECHANISMS INVOLVED IN SPLENOPATHIES.[1]

By C. R. B. BLACKBURN, M.D.,
Sydney.

A PHYSICIAN cannot be expected to speak profoundly on pathology even though he speaks for the Section on Pathology. Were I to do so, you would surely say: *"C'est la profonde ignorance qui inspire le ton dogmatique."* (La Bruyère.) I will try to avoid this by discussing the pathological and physiological mechanisms involved in some of the splenopathies which may be amenable to treatment by splenectomy. I make no pretence of mentioning all, or even most, of the indications for splenectomy, or of discussing the general effects of splenectomy.

It is necessary briefly to indicate two functions of the spleen. These functions are evident in the appropriate physiological or pathological circumstances.

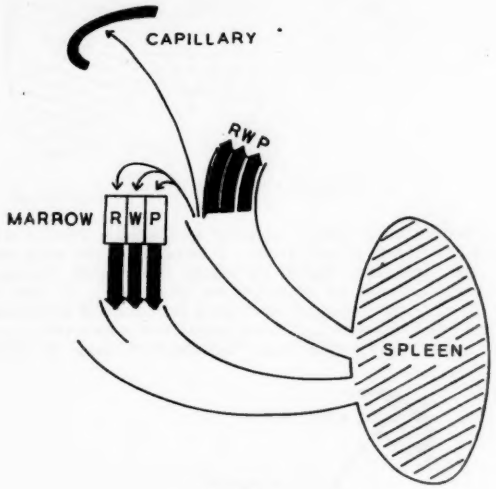

FIGURE I.
Normal relations.

1. The spleen can remove certain red blood cells from the blood passing through it—this especially applies to spherical and aged cells. In diseased spleens there may be an increased removal of red blood cells.

2. The spleen appears to have a function as "hormone" producer—it can produce effects on distant organs and must do so by some chemical means. For the sake of discussion I will postulate that the spleen normally produces a substance (or substances) "X". "X" is carried in the splenic venous blood to the portal vein and thence to the liver, where it may be altered or partly destroyed. "X" may then enter the general circulation to be conveyed to the body generally. The effects of this substance or substances are recognized, in splenopathies, by an inhibitory effect on the bone marrow and an increased capillary fragility. The reverse effects may be seen after splenectomy. Of course, it is possible that the spleen produces a substance which is effective in causing the liver to produce (or to inhibit the hepatic production of) a substance which has the general properties already ascribed to the splenic substance "X". This concept that the spleen exerts an effect on the bone marrow and on capillary fragility is essential to the understanding of the underlying mechanisms of which I am to speak. I will make passing reference to some other functions of the spleen a little later on.

In Figure I there is a schematic representation of the major points mentioned. The bone marrow is shown pouring red blood cells, white blood cells and platelets into the blood-stream which enters the splenic artery. The blood is conveyed to the spleen and leaves by the splenic vein. Blood from the splenic vein is shown to *"feed back"* to the marrow, where the "X" substance tends to exert an inhibitory effect. I have also shown this same substance exerting an influence on the capillaries. This simple diagram is not intended to be all-embracing, but to serve as a scaffolding for the subsequent remarks on diseased spleens.

Idiopathic Thrombocytopenic Purpura.

Idiopathic thrombocytopenic purpura is perhaps the simplest abnormality of the spleen appearing to produce functional disturbances reversible by splenectomy. Dameshek and others (1946) have reported that examination of the bone marrow, by biopsy, reveals diminished production of platelets from apparently normal megakaryocytes. This is difficult to demonstrate, and other investigators have failed to do so. I do not think these findings are very useful—the significant finding is the presence of megakaryocytes in a marrow without evidence of other disease.

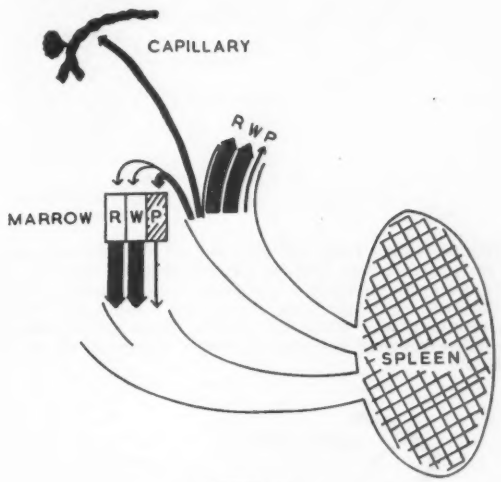

FIGURE II.
Idiopathic thrombocytopenic purpura.

Purpura is due to abnormal capillary fragility.

The mechanism is illustrated in Figure II. The spleen is functioning abnormally, as it appears to be producing an abnormal substance (or an excess of a normal substance) which has an inhibitory effect on the bone marrow and especially on the megakaryocytes. The outstanding demonstrable effect of this inhibition is a diminished number of platelets in the peripheral circulation, thrombocytopenia, apparently due to inadequate production by megakaryocytes in the bone marrow. The capillary defect is shown as directly related to the spleen, though there is no proof of this.

Clearly splenectomy should, and usually does, cure this condition. Failure to cure may be attributed to the following two causes. (i) Failure to remove accessory splenic tissue. Accessory spleens are very common and are practically always found in the splenic hilum or along the splenic vein. (ii) Mistaken diagnosis. Thrombocytopenic purpura is not necessarily an indication for splenectomy, and usually is not; but idiopathic thrombocytopenic purpura usually is an indication. Idiopathic thrombocytopenic purpura implies no determinable cause for the condition, typical bone marrow findings, and characteristic hæmatological and capillary findings in the appropriate patient.

There appears to be a correlation between time of operation and intracranial bleeding in idiopathic thrombocytopenic purpura—the longer the delay, the greater the likelihood of this occurrence. Idiopathic thrombocytopenic

purpura is treated as an emergency in Doan's and Damashek's clinics, where there is a very low incidence of intracranial bleeding. Others, who tend to wait for some time before operation, have incidences of up to 12% (all bleeding within the skull is included, and not only major cerebral hæmorrhage).

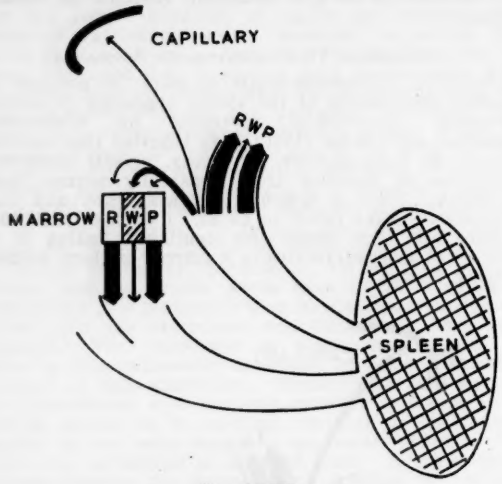

Figure III.
Splenic neutropenia.

The mortality from splenectomy in this disease should be no greater than for an appendicectomy when a practised surgeon operates. I suggest that females admitted to hospital with purpura should be treated as "medical emergencies" and when an accurate diagnosis of idiopathic thrombocytopenic purpura is made, regarded as surgical patients.

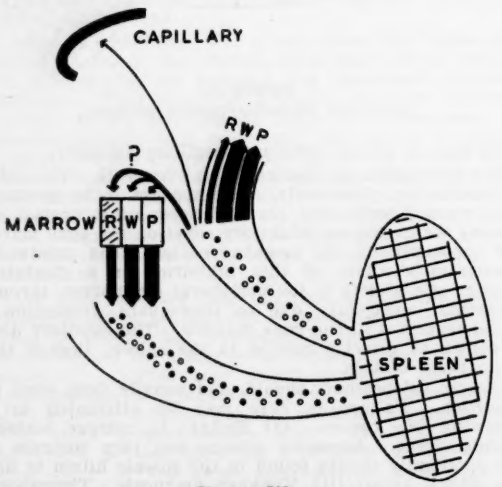

Figure IV.
Congenital hæmolytic icterus.

Primary Splenic Neutropenia.

Primary splenic neutropenia (Rogers and Hall, 1945) is rather similar to idiopathic thrombocytopenic purpura, except that the neutrophile cells are affected and there is no capillary defect. The similarity of the basic mechanisms is seen by a comparison of Figure II and III. There is an inadequate delivery of myeloid cells from the marrow to the blood-stream.

It must be mentioned that there are reports of splenic neutropenia associated with an abnormal avidity of the spleen for neutrophile cells; it could be shown in Figure III by a normal arrow in from the marrow and a small arrow out of the spleen. The diagnosis in this type of case depends on the demonstration of a pronounced difference between the splenic arterial and splenic venous white blood cell count at operation. I have seen one such case; but the evidence, based on white blood cell counts at operation, cannot be regarded as conclusive. If this was the whole story, one would expect pronounced hyperplasia of the bone marrow and some immature white cells in the circulation. Hyperplasia of the marrow may be found; but immature cells must be quite exceptional. Furthermore, a hyperplastic marrow does not necessarily indicate the delivery of a large number of cells to the peripheral circulation.

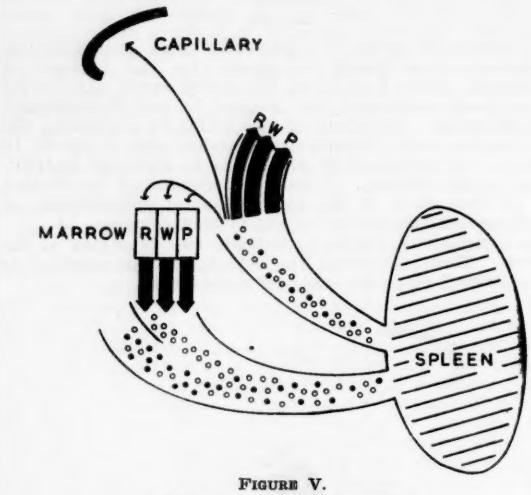

Figure V.
Acquired hæmolytic icterus.

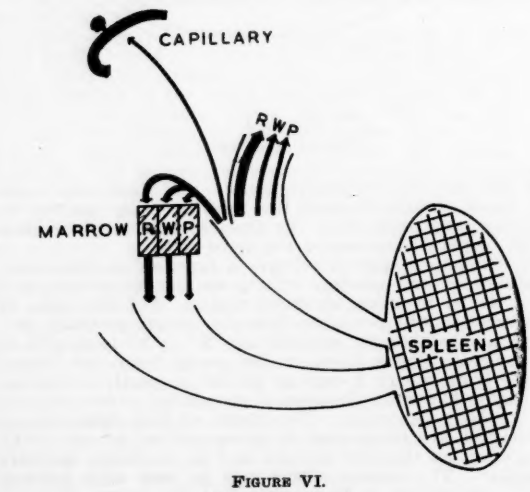

Figure VI.
"Hypersplenism."

Primary splenic neutropenia is usually associated with normal or hyperplastic myeloid tissue in the bone marrow. Splenomegaly is the rule, a history of frequent infections common, and remarkable remission follows splenectomy.

I would remind you that a guarded prognosis must be given to these patients, for splenectomy is followed by

temporary leucocytosis in normal subjects as well as in most patients. A temporary remission may be followed by recurrence and ultimate death. In these circumstances, if there is no accessory splenic tissue, the diagnosis of primary splenic neutropenia was mistaken. Some other tissue may possibly take over the abnormal function of the removed spleen, but there is little evidence of this at the present time. I would include Felty's syndrome in the general category of splenic neutropenia.

Congenital Hæmolytic Icterus.

Congenital hæmolytic icterus is a somewhat more complicated disease. The mechanism is illustrated in Figure IV, in which the bone marrow is shown producing abnormal red cells (the solid black circles) which are the typical "spherocytes". These abnormal red cells are preferentially removed by the spleen (Young, 1947)—not completely in one cycle, but nevertheless at a much greater rate than are the normal red cells. In the diagram the red cell tomy would be expected to produce a diminished rate of

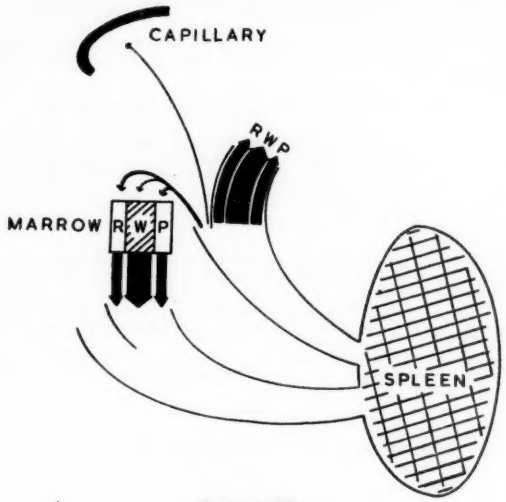

FIGURE VII.
Leuchæmia.

red-cell destruction, as in congenital hæmolytic icterus, but that is all. The basic mechanism of the disease remains arrow leaving the splenic vein is smaller than the one entering the splenic artery from the bone marrow. Anæmia is present.

An increased inhibitory effect from the spleen on the bone marrow resulting in a diminished production of red cells, white cells and platelets is also illustrated. The question mark implies some doubt regarding this. Owren (1948) has produced good evidence that crises in congenital hæmolytic icterus are associated with temporary hypoplasia or aplasia of the bone marrow. There could then be a normally functioning spleen removing spherical cells from the blood passing through it, and splenectomy would simply lengthen the life of the abnormal red cells. Subsequent temporary hypoplastic marrow episodes would fail to produce a clinical crisis, because the red cells would live long enough to prevent a profound fall in their number in the circulation. On the other hand, the marrow changes could well be due to cyclic inhibitory effects from the spleen (Damashek and Bloom, 1949). Splenectomy would then result in complete cure of the patient. Repeated marrow examination after complete splenectomy should indicate which mechanism is involved.

Acquired Hæmolytic Icterus.

Acquired hæmolytic icterus is based on a somewhat different mechanism. As is shown in Figure V, the red cells are considered to be relatively normal on leaving the marrow, but become altered by adsorption of an auto-agglutinin in the circulation. These "coated cells" commonly give a positive response to Coombes's test. In Figure V the mechanism is somewhat simplified, of necessity, for all these patients have not precisely the same mechanism for their disease.

First consider the simple story as shown. Coated red cells, often spherical, tend to be removed during their passage through the spleen and anæmia tends to develop. A hyperplastic marrow attempts to compensate. Splenec- unaffected. More and more cells may become agglutinable, and tissues other than the spleen help in the excessive destruction of these cells.

In many of these patients there may also be an inhibitory effect on the bone marrow from the enlarged spleen, much as shown in Figure IV. When this is the case, splenectomy would have the additional advantage of "releasing" the bone marrow inhibition, and more benefit may follow than could be attributed to the increased life span of the red blood cells.

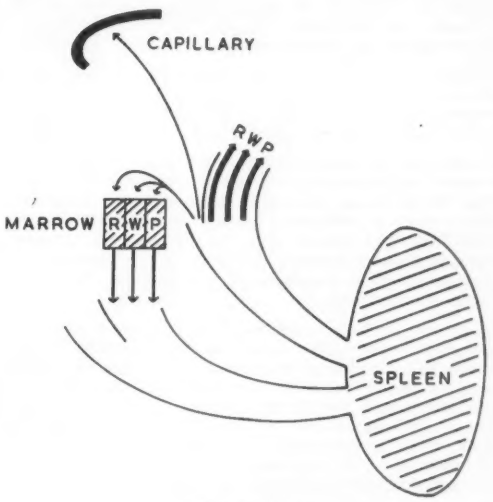

FIGURE VIII.
Myelofibrosis.

Gardner (1949) has shown that, in the appropriate circumstances, red cells incubated with spleen from patients giving a positive response to the Coombs test become covered with antibody and give a positive response to Coombs's test. Clearly if the spleen was the only site of antibody production complete cure might be expected to follow splenectomy. Unhappily such is not usually the case—the antibody is not solely produced in the spleen.

The response to complete splenectomy in acquired hæmolytic icterus will therefore depend on the degree to which the following factors are operating in the production of the anæmia: (i) the removal of the abnormal red cells by the spleen; (ii) the degree of marrow inhibition by the enlarged spleen; (iii) the amount of antibody produced by the spleen.

Hypersplenism.

In the conditions mentioned there has been continued reference to the inhibitory effect of the spleen on bone marrow—usually referring to the development of one or more of the blood-cell types. These are all instances of so-called hypersplenism.

In Figure VI the major effects of an over-active spleen are illustrated. There is bone marrow inhibition, together with, in some instances, a capillary effect leading to petechial hæmorrhage function. Clearly splenectomy should be of great benefit to patients in whom a mechanism such as this is in operation, the degree of temporary

or permanent benefit depending on the cause of the over-activity of the spleen.

The causes of hypersplenism are usually classified as primary or secondary (Doan and Wright, 1946). No attempt will be made to present a complete list of these, but reference will be made to some of them. Figure VII illustrates that hypersplenism may be a factor in leuchæmia—a tendency towards a depression of red-cell and platelet production and delivery to the circulation. Splenectomy in an occasional case of chronic leuchæmia has proved of benefit, for there has been less depression of red-cell formation, platelet counts have become more normal and there may be less purpura (not illustrated in Figure VII).

The mechanism illustrated in Figure VI is important in many diseases. In Hodgkin's disease there may be an associated hæmolytic anæmia with a positive response to Coombs's test as well. Many chronic infections such as malaria, tuberculosis and kala-azar (Cartwright *et alii*, 1948) provide evidence of an over-active spleen. In Gaucher's disease a profound marrow depression may promptly be relieved by splenectomy. In certain of the reticuloses an additional factor in the production off anæmia may be an active splenic erythrophagocytosis.

Splenectomy may then be indicated in any of these conditions in certain cases. In many disorders the marrow depression is a more urgent indication for splenectomy than are the inconvenience and danger of a huge spleen.

In contrast to hypersplenism, patients with myelofibrosis and some patients with so-called "aplastic anæmia", in whom there is an extreme depression of bone-marrow function, may be killed by splenectomy. As illustrated in Figure VIII, the spleen may be the only significant site of extramedullary hæmatopoiesis, and the patients depend on this production for their very existence. It is fatal to perform splenectomy.

To return to hypersplenism and Figure VI: a common cause of an over-active spleen is the so-called congestive splenomegaly—a term used here to cover any cause of congestion and including cirrhosis of the liver, portal hypertension and Banti's syndrome. As a rule the anæmia in these patients is due to hæmorrhage from œsophageal varices, from hæmorrhoids or from peptic ulceration, but there may also be depression of erythropoiesis. Leucopenia and thrombocytopenia are characteristic.

Splenectomy can be relied upon to relieve bone-marrow depression, but is frequently contraindicated except as a part of an anastomotic operation. The treatment of this condition depends on the cause, upon the site of venous obstruction, and upon whether one attempts to relieve the obstruction or to save a life, or to do both. Obstruction of the splenic vein can well be treated by splenectomy, whether it is due to hydatid disease, post-traumatic thrombosis or aneurysm *et cetera*. Obstruction of the portal vein is better treated by splenectomy followed by anastomosis of splenic and renal veins, or of portal vein to inferior *vena cava* (Whipple, 1946; Blakemore, 1947). Simple splenectomy is of value when anastomotic procedures are technically impossible, for it reduces the portal vein inflow by some 25% to 40% (Whipple, 1945). Relief of bone marrow depression and some temporary relief of the portal hypertension will follow. When anastomosis is impracticable, gastrectomy, partial or total, and œsophagectomy (Phemister and Humphreys, 1947) may save the life and relieve the symptoms of the patient having repeated hæmatemeses after splenectomy.

Banti, who described both the anæmia and the degenerative changes in the splenic vein in splenic anæmia, does not seem to have recognized the raised portal pressure. In 1898 he said: "*Sublata causa tollitur effectus*" (Banti, 1937)—"By removal of cause the effect is removed", and he considered that splenectomy was urgently to be recommended. We agree with his reasoning, but knowing more of the causes and mechanism, differ from his recommendation for splenectomy. Yet "*malum consilium est, quod mutari non potest*" (Publius Syrus)—applicable to our present discussion as much as to Banti.

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INDICATIONS FOR SPLENECTOMY.[1]

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Sydney.

Physiology.

In ancient days the adherents of the humoral theory believed the spleen to be a source of the black bile—regarded by some as a cold dry humour and by others as a hot wet one. The spleen has been regarded as the seat of anger and hatred; hence the common, as distinct from the anatomical, use of the term today.

Tennyson wrote:

> . . . He chew'd
> The thrice-turn'd cud of wrath, and cook'd his spleen.

And in Pope's translation of the *Iliad* we read:

> Spleen to mankind his envious heart possess'd,
> And much he hated all, but most the best.

Matthew Green came nearer to physiological truth than he knew when he wrote:

> To cure the mind's bias, Spleen,
> Some recommend the bowling green;
> Some, hilly walks; all, exercise;
> Fling but a stone, the giant dies.

Within recent years our knowledge of the spleen has increased greatly. But still it is in many ways a mysterious organ.

Several functions are well known; some others are assumed. An attractive field for further research is presented.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on July 27, 1950.

The spleen serves as a reservoir of blood. It contains a large amount of blood, and this blood is richer in erythrocytes than the circulating blood in the proportion of 3:2. When the supply of oxygen to the tissues falls below their requirements, the capsule of the spleen contracts and squeezes the rich blood out. The spleen diminishes in size in the stress of exertion or emotion. Could it be that the ancient idea of the spleen as a seat of the emotions was founded on observed fact? It has been said that the ancients would remove the spleen from a long-distance runner to increase his stamina. If this is true, one would think that there should have been a serious shortage of long-distance runners: first, because of the reluctance to suffer mutilation, and secondly because of the high mortality rate that would have accompanied such an operation.

The spleen has been termed the "graveyard of the erythrocytes". Probably the greatest destruction of red blood cells occurs in the general circulation and is purely traumatic, the cells most vulnerable to injury being the older cells. But many of the cells that have completed their term of useful existence are carried to the quiet pools of the splenic sinuses, where they end their days in peace. It is not known how they die. Some physiologists believe that they are taken up by the macrophages of the splenic pulp. Best and Taylor (1940) paint a grim picture:

> . . . all the red cells in the body are brought into direct relationship with the splenic tissues to pass inspection by the macrophages lurking in the pulp and lining the sinuses. To these cells the infirm, senile or dying erythrocytes fall a prey.

Dameshek (1947) does not agree with this idea. While he says that the means of destruction is unknown, he points out that the red cells are made thicker by stasis and that thick cells are more vulnerable. He suggests that the destructive agent may be chemical, and he mentions lysolecithin. The weight of evidence seems to suggest that the macrophages ingest debris only.

In the embryo the spleen is an important factory for all the elements of the blood. Normally in adult life it manufactures only monocytes and lymphocytes; but it remains a potential blood-forming organ and it may exert this function at times of stress.

Some other functions attributed to the spleen are as follows: (i) It either destroys thrombocytes or inhibits their excessive production. (ii) It either destroys leucocytes or inhibits their excessive production. (iii) It exerts some control on the denucleation of red cells and on their emission from the marrow. (iv) It exerts some control on the thickness of red cells (it makes them thicker). (v) It acts as lymphoid tissue in providing a filter in infections. (vi) It is concerned in the production of bilirubin. In this regard it has been noted that the splenic vein contains more bilirubin than the splenic artery. (vii) It is a storehouse of iron.

Dameshek postulates the existence of splenic hormones, which regulate the development of cells and their emergence from the blood-forming organs. This is an attractive theory. But the phenomena could be accounted for by a destructive effect.

Doan and Wright (1949) wrote as follows:

> The spleen is the principal organ for clearing the blood stream of debris, including damaged, sensitized, debilitated and senile blood elements. The splenic circulation is ideal for the physiologic stagnation of the blood stream, with a corresponding sequestration and concentration of the formed elements as the plasma continues its circulation.

King (1914) suggested that this function of the spleen might get out of hand; that the spleen might fail to discriminate between senile and normal cells. To this state of abnormal or excessive splenic activity he applied the term "hypersplenism". Within recent years the use of this term has been revived. Considerable evidence has accumulated in support of King's hypothesis.

The Effects of Splenectomy.

The spleen is not essential to life. On its removal many of its functions are taken over by other parts of the reticulo-endothelial system.

On removal of the spleen of a normal person all the cellular elements of the blood increase in numbers. In some cases a transient anæmia occurs. The increase in numbers of red cells is not great—perhaps about half a million per cubic millimetre. The numbers of target cells are increased, and the fragility of the red cells is diminished. Howell-Jolly bodies are common. Their presence is thought to indicate some abnormality in the extrusion of the nucleus. The destruction of red cells is diminished.

The leucocytes increase greatly in numbers. Counts of 20,000 to 40,000 per cubic millimetre are common. Numerous young forms are seen. A perusal of the literature may leave one in doubt whether the leucocytosis is general or whether the increase is mainly in the polymorphonuclear cells or in the lymphocytes. Probably what happens is that the polymorphonuclear cells increase immediately after splenectomy as after any other operation of like severity. Later the increase is mainly in the lymphocytes.

The leucocytosis usually subsides in a few weeks, but may continue much longer. One patient at Sydney Hospital still has a leucocyte count of 15,000 per cubic millimetre, twelve months after splenectomy.

The platelets increase in number to perhaps over 1,000,000 per cubic millimetre. The count may remain above normal for months.

The reserve supply of iron is reduced.

Valuable knowledge has been gained from a study of the results of splenectomy. But it does not make it clear whether the activity of the spleen is mainly inhibitory on the marrow or mainly destructive of the cells. However, as a result of these observations the indications for splenectomy are becoming more clearly defined.

At present we seem to be on the crest of the splenectomy wave. There has been perhaps a too enthusiastic acceptance of Dameshek's views. And, as invariably happens when some spectacular therapeutic measure is introduced, splenectomy has been misused.

Splenectomy in skilled hands is not a very dangerous operation. Since 1922, splenectomy has been performed on 32 occasions at Sydney Hospital. Only two patients have died after splenectomy, and these were patients suffering from rupture of the spleen.

TABLE I.

Splenectomy at Sydney Hospital (1922 to 1950).

Diagnosis.	Number of Cases.	Deaths.
Rupture of spleen	12	2
Acholuric jaundice	6	—
Banti's disease	7	—
Thrombocytopenic purpura	6	—
Aplastic anæmia (?)	1	—
Total	32	2

Hypersplenism.

The term "hypersplenism" is assumed to mean excessive activity in one or more of the spleen's functions. It may be manifested by a reduction in the numbers of any or all of the three main types of cells in the circulating blood. Leucocytopenia, erythrocytopenia, thrombocytopenia or "panhæmatocytopenia" may occur. In addition, the spleen may cause hæmolysis—probably by phagocytosis.

Hypersplenism may occur apparently idiopathically; it may occur as a part of some disease of the hæmatopoietic system, or in the course of some generalized infection; and it may occur when the spleen is attacked by primary or metastatic neoplasm.

Therefore, hypersplenism may occur in such disorders as leuchæmia, Hodgkin's disease, rheumatoid arthritis (Felty's syndrome), syphilis, miliary tuberculosis and malaria; it may be merely an incidental feature or it may be the main feature of the illness. When it appears incidentally, its

effects may be so damaging as to have a profound influence on the course of the disease and to threaten the patient's life.

The diagnosis of hypersplenism depends on the history and physical examination, on examination of the peripheral blood and bone marrow, and perhaps on the results of the adrenaline test. In the physical examination it is well to bear in mind that splenic hypertrophy may be considerable before it can be demonstrated clinically. Examination of the bone marrow is essential, as the diagnosis of hypersplenism is not to be made unless there is a compensatory hyperplasia of the cells that are deficient in the blood. On the other hand, if the marrow is hypoplastic in the presence of splenomegaly, it is probable that the spleen is exercising its primeval function of hæmatopoiesis. If such a spleen was removed, the patient would be worse off. In this regard, a case reported by Doan and Wright (1949) is of interest.

The patient was a woman, aged thirty-eight years, who had been exposed to benzol in industry. She had panhæmatocytopenia. Her bone marrow was hypoplastic. The spleen was not enlarged. Injection of adrenaline evoked a transitory increase in the cells of the blood. Splenectomy was advised with the object of removing the splenic reservoir and releasing the "full hæmatopoietic function potential of the marrow". The blood picture returned to normal and remained so.

The adrenaline test consists in the subcutaneous injection of adrenaline and the making of a blood count at intervals of twenty minutes or half an hour over a period of several hours. The adrenaline causes contraction of the spleen and issue of the splenic blood into the circulation. If a pronounced rise in the red cell count occurs, it may be taken that the spleen is exerting an abnormal function in the storage of these cells. Dameshek regards the test as useless. He states that a positive result may be obtained after splenectomy.

Indications and Contraindications.

Diseases in which splenectomy may and may not be of value are shown in Table II.

Rupture.

Since 1922, 17 patients have been treated at Sydney Hospital for rupture of the spleen. Splenectomy was performed in 12 cases. Two patients died after splenectomy. In all these cases the spleen had ruptured as a result of severe injury, such as a fall from a height on to a paling fence. But if the spleen is large and soft as a result of infection, it may rupture as the result of quite a slight blow. Indeed, it has been recorded that a man ruptured his spleen merely by jumping from a wagon to the ground. Many an inexperienced overseer has been charged with manslaughter for giving a native employee a prod in the belly. The native appears to be lazy or slow to obey a command. This is because he is ill with malaria. The overseer regards him as recalcitrant, and instead of treating him for his illness, he administers corporal punishment, with a fatal result.

Rupture of the spleen is, of course, the most urgent indication for splenectomy.

Acholuric Jaundice.

It is perhaps in familial acholuric jaundice that splenectomy has its most strikingly beneficial effects. It is an essential part of the treatment in most cases. But due attention must be paid to the age of the patient and the degree of anæmia before splenectomy is decided upon. Some people go through life with spherocytosis, but with little, if any, hæmolysis. In some people the disease does not become evident until middle life or later, and then it may consist of nothing more than an occasional mild hæmolytic episode. Such people may have very little disability. Operation for them may be considered unnecessary. But even in these people a sudden severe hæmolytic paroxysm may occur.

The rule is to perform splenectomy on all affected children or young adults, however mild the symptoms, and on older persons if the symptoms are severe.

Regular examination should be made of any patient who is not subjected to splenectomy.

In some cases the difficulty is in the choice of a suitable time to operate. Some patients are subject to hæmolytic crises. Operation at such times should be avoided as far as possible.

Blood transfusion should be avoided. Doan and Wright mention that, if adrenaline is injected into the spleen after the splenic artery is tied and before the vein is tied, as many as 2,000,000 red cells per cubic centimetre may pour into the circulation. "Actually", they state, "preoperative transfusion is contraindicated in that it frequently appears to add fuel to an already extensive conflagration of cellular destruction."

TABLE II.

Indications For and Against Splenectomy.

Necessary.	Of Possible Value.	Valueless.
(i) Rupture.[1]	(i) Mediterranean and sickle cell anæmia (if the accent is on hæmolysis).	(i) Sclerosis of marrow with compensatory hypertrophy of spleen.
(ii) Acute idiopathic thrombocytopenic purpura.[1]	(ii) Rare cases of leuchæmia and lymphoma with splenic hæmolysis.	(ii) Leuchæmia and lymphoma.[2]
(iii) Idiopathic thrombocytopenic purpura.[2]	(iii) Splenomegaly that is so great as to interfere with locomotion or to be a source of danger because of the liability to rupture on slight trauma.	(iii) Mediterranean and sickle cell anæmia.[2]
(iv) Acute hæmolytic anæmia.	(iv) Miliary tuberculosis.	(iv) Secondary thrombocytopenia.
(v) Familial acholuric jaundice (spherocytosis).		(v) Malaria.
(vi) Splenic hæmatocytopenia.		(vi) Kala azar.
(vii) Hypersplenism secondary to rheumatoid arthritis, Gaucher's disease, chronic congestive splenomegaly *et cetera*.		
(viii) Cysts, tumours and abscesses of the spleen.		

[1] Urgent.
[2] In most cases.

On September 12, 1947, a girl, aged eleven years, gave a history that since the age of two years her urine frequently had been dark. At the age of nine years she commenced to have diarrhœa; the stools became very loose and frequent, and sometimes they contained blood. She got some relief from sulphaguanidine. She had been told that she had acholuric jaundice. Neither of her parents nor her brother, aged sixteen years, had any symptoms of acholuric jaundice. Her father had been in New Guinea during the war and had had dysentery on several occasions.

The child was tall, slender, emaciated and exceedingly pale. The liver was palpable. The lower border of the spleen was almost at the level of the iliac crest. The hæmoglobin value was 5·0 grammes per 100 millilitres. On this first day a full blood count was not made. No specific cause for the dysentery was discovered.

Later, spherocytes were found in the blood. The red cell fragility was increased. During the next four months the diarrhœa subsided considerably under symptomatic treatment. On a number of occasions she suffered from hæmolytic crises of greater or less severity, during which petechiæ appeared and effusion of blood occurred in a joint and once into the orbit.

In January, 1948, the hæmoglobin value was 8·5 grammes per 100 millilitres, the erythrocytes numbered 2,930,000 per cubic millimetre, and the leucocytes 4600 per cubic millimetre.

Dr. Howard Bullock performed splenectomy on January 17, 1948. No blood transfusion was given. The child lost very little blood at the operation and she made a satisfactory and rapid recovery. Her colour improved rapidly. On February 2 her hæmoglobin value was 11·9 grammes

per 100 millilitres, and the red cells numbered 3,890,000, the leucocytes 11,200 and the thrombocytes 1,360,000 per cubic millimetre. The red cells were still abnormally fragile *in vitro*.

She has grown into a strong, plump, healthy looking girl. Her latest blood count, made on March 13, 1950, reads as follows: hæmoglobin value, 11·8 grammes *per centum*; red cells, 4,760,000 per cubic millimetre; hæmatocrit reading, 38; mean corpuscular volume, 79 cubic micromillimetres; mean corpuscular hæmoglobin, 25 micromicrogrammes; mean corpuscular hæmoglobin concentration, 31%; leucocytes, 8200 per cubic millimetre; polymorphonuclear cells, 69%; eosinophile cells, 3%; lymphocytes, 22%; monocytes, 6%; platelets, 440,000 per cubic millimetre. Many of the red cells are spherocytes. The anæmia is orthocytic orthochromic. Howell-Jolly bodies are present in the red cells. A fragility test shows that hæmolysis commences in 0·6% sodium chloride solution and is complete in 0·4%. The bleeding time is one minute and three-quarters. The coagulation time is five and a quarter minutes (normal, four to seven).

Her bowels still tend to be loose and have to be kept in check with bismuth. It will be noted that only the splenic influence has been removed .The inherent defect remains.

Since 1922 acholuric jaundice has been diagnosed in 15 cases at Sydney Hospital. Six patients have been subjected to splenectomy, with satisfactory results. In one other case, the patient was admitted to hospital with rupture of the spleen and was found to have spherocytosis and other evidence of familial acholuric jaundice.

Hæmolytic Anæmia.

The case summarized below exemplifies the kind of problem that the physician may encounter. In this case splenectomy failed. It may have hastened death. However, what seems more likely is that the introduction of foreign material (blood) was the main factor in the patient's downfall.

A man, aged forty-seven years, a pensioner, was admitted to the Royal South Sydney Hospital on June 24, 1948, complaining of loss of weight, hæmoptysis, weakness and breathlessness. He was found to have a pleural effusion, with probably an infective cause. He had a polymorphonuclear leucocytosis and a grave anæmia, with macrocytosis and a colour index of 1·0. Examination of the gastric contents revealed achlorhydria. He was treated for pernicious anæmia, but he proved to be allergic to liver administered parenterally. As he recovered from his pleural infection his leucocyte count fell. On July 27 his hæmoglobin value was 10·1 grammes per 100 millilitres, and his red cells numbered 2,620,000 and leucocytes 2900 per cubic millimetre. Platelets were scanty. He was discharged from hospital on August 5, 1948, and was directed to attend the out-patient department.

Examination of the sternal marrow showed megaloblastic changes.

He did not benefit from folic acid. He was given proteolysed liver by mouth. Attempts were made to desensitize him to liver extracts, but without avail. Purpura appeared when liver extract was given parenterally. The physician looking after him as an out-patient managed to keep him moderately well, with a red cell count varying from about 2,500,000 to 3,000,000 per cubic millimetre. He was given a blood transfusion in February and again in March, 1949, with temporary relief. He was admitted to hospital again on September 10, 1949, by which time his spleen could be felt about one inch below the costal margin.

Here was a man with splenomegaly and panhæmatocytopenia. He was unable to take liver extract parenterally or orally. Folic acid did him no good. He had hæmorrhagic tendencies. He had no evidence of subacute combined degeneration of the spinal cord. The choice seemed to be between splenectomy and repeated blood transfusion. Splenectomy might fail; but if it was effective he would have a chance of leading a reasonably comfortable life. Splenectomy was performed. At operation a large extravasation of blood was seen under the peritoneum of the small intestine. A blood transfusion was given.

From the time of operation he went downhill. He became intensely jaundiced. His leucocyte count rose; but his red cell count fell, and platelets were still scanty. He died five weeks after operation.

An interesting but probably insignificant feature was the presence of ovalocytes in his blood. This was found to be a familial trait.

Thrombocytopenia.

Splenectomy is indicated in idiopathic thrombocytopenic purpura, but not in secondary thrombocytopenic purpura.

Since 1922, 33 patients with thrombocytopenic purpura have been admitted to Sydney Hospital. Unfortunately, the cases are not listed under the headings of "idiopathic" and "secondary". Splenectomy was performed on six patients. The results were variable. No deaths occurred. It is doubtful whether the following case was one of idiopathic thrombocytopenia.

The patient was a man, aged fifty-three years, who gave a history of hæmorrhage and purpura. He was admitted to hospital in February, operated on in April, and discharged in June, 1938. His record is shown in Figure I. It will be

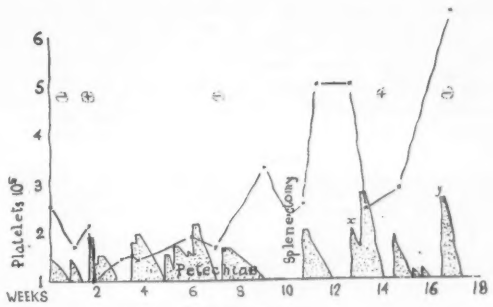

FIGURE I.

"+", petechiæ from tourniquet test; "–", no petechiæ from tourniquet test; "x", allowed up; "y", walked a great deal.

noted that the platelets increased in number after splenectomy, but that he continued to have large numbers of petechiæ. Some factor other than thrombocytopenia seems to have been operating here.

In other cases the response to splenectomy has been dramatic.

Banti's Syndrome (Chronic Congestive Splenomegaly).

Since 1922, 45 patients with Banti's disease, splenic anæmia or splenomegaly, have been admitted to Sydney Hospital. Splenectomy was performed in seven cases. On the whole, the results of operation in this series can be regarded as satisfactory. No fatality occurred. The following case may be taken as an example. Splenectomy saved the patient's life.

The patient was a woman, aged thirty-six years, who was admitted to hospital on November 30, 1944, complaining of weakness and breathlessness of twelve months' duration. She had had jaundice twice and hæmatemesis on several occasions. She was found to be pale and slightly yellow. She was gravely anæmic and she had leucopenia and thrombocytopenia. The hæmoglobin value was 5·2 grammes per 100 millilitres; the red cells numbered 1,870,000 and the white cells 3100 per cubic millimetre. Repeated blood transfusion served merely to keep her alive. Splenectomy was performed on March 14, 1945. No further blood transfusion was required. At the time of her discharge from hospital on April 8, 1945, her hæmoglobin value was 13·1 grammes per 100 millilitres; her red cells numbered 4,390,000 and her leucocytes 7050 per cubic millimetre; platelets were plentiful.

The result of the Kahn test in this case was "++++". Perhaps syphilis coloured the picture. Perhaps she had syphilis of the spleen.

Aplastic Anæmia.

Splenectomy is contraindicated in aplastic anæmia. It is mentioned here because of an unusual case in the Sydney Hospital series. (See Figure II.)

The patient was a man, aged thirty-four years, who had been anæmic since childhood. He gave no history of exposure to benzol or other material known to be toxic to the marrow. During the six months prior to admission to hospital in May, 1949, he had been given blood transfusion a number of times. Transfusion was again neces-

sary on several occasions before splenectomy was performed one month after his admission to hospital. At the time of his admission his hæmoglobin value was 6·8 grammes per 100 millilitres of blood; the red cells numbered 2,220,000 and the leucocytes 3200 per cubic millimetre. Macrocytes and occasional stippled cells were seen. Platelets were plentiful. Later it was noted that the platelets, though plentiful, were abnormal. The cellularity of the marrow was said to be normal.

After the operation the lower lobe of his left lung collapsed, and a pleural effusion appeared. This considerably delayed his recovery. At the end of July his hæmoglobin

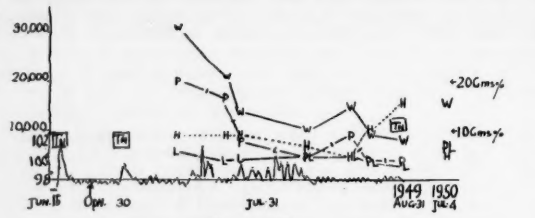

Figure II.

"W", total leucocytes per cubic millimetre; "P", neutrophile leucocytes; "L", lymphocytes; "H", hæmoglobin value in grammes *per centum*; "Tn", transfusion.

value was 9·7 grammes per 100 millilitres; his leucocytes numbered 21,000 and red cells 2,800,000 per cubic millimetre; platelets were very numerous.

Since his discharge from hospital in October, 1949, he has been readmitted twice for blood transfusion, the last time in April, 1950. On July 4, 1950, his hæmoglobin value was 5·7 grammes per 100 millilitres; his red cells numbered 3,680,000 and his leucocytes 15,300 per cubic millimetre. Platelets were plentiful. Numerous Jolly bodies were seen. Numerous normoblasts, macrocytes and anisocytes were seen.

The diagnosis of aplastic anæmia was made in this case, probably for want of a better. But the case does not conform to the usual description of aplastic anæmia. Splenectomy seems to have done some good. He has not required such frequent blood transfusion since the operation.

Malaria, Tuberculosis and Syphilis.

In malaria the spleen becomes swollen, but returns to normal or nearly normal on treatment. But in some people who have suffered repeatedly from malaria over many years and have received inadequate treatment the spleen may be permanently enlarged and may be huge. In such a case no doubt persistent hypersplenism could occur. But it must be very rare. Leucopenia and severe anæmia are common in inadequately treated malaria. Whatever the size of the spleen, and whatever the degree of anæmia, in my experience, the blood recovers with appropriate antimalarial measures and the administration of iron.

In the case recorded below, Banti's disease was tentatively diagnosed when the patient was first examined.

The patient, a man, aged twenty-four years, sought medical advice on November 18, 1924. He was born in Queensland, but had lived in the Bainings district of New Britain since the age of five years. His living conditions were not up to European standards.

He complained of lassitude since an attack of dysentery six weeks earlier. He said that his belly felt tight. He said that he had not had much malaria. He was a strongly built man, but very pale. His spleen filled the left flank. It extended below the iliac crest and to the right beyond the mid-line of the abdomen; the notch was palpable just above the umbilicus. The leucocytes numbered 3800 and the red cells 3,000,000 per cubic millimetre. Poikilocytosis, anisocytosis and an occasional nucleated red cell were seen. He was given quinine. He declined admission to hospital. A week or so later he went home. He reported again on December 13, stating that he felt quite well. The spleen was considerably reduced in size.

On September 13, 1925, he reported again. He felt quite well and he looked well. The lower border of the spleen was now one hand's breadth below the costal margin. The medial border was in the epigastrium. The leucocytes numbered 5700 and the red cells 6,000,000 per cubic millimetre.

In my experience the only malarial spleen demanding removal is the ruptured one, and as a rule the only opportunity for removing that is in the post-mortem room. Bursting of the large, soft spleen of a patient with untreated malaria causes bleeding that is terrific and rapidly fatal.

Blackwater fever is a complication of malaria. In this grave disease the spleen is always hypertrophied and blood destruction is great. Is this hæmolysis due to some devilish activity of the spleen?

Hypersplenism may occur in miliary tuberculosis. The physician will have to make up his mind whether, in view of the gravity of the disease, splenectomy would be justified; whether the cure of hypersplenism is likely to help the patient to recover.

In hypersplenism of syphilis, a sane rule is first to treat the syphilis thoroughly. If hypersplenism persists to the detriment of the patient's general health, there is nothing to be lost by splenectomy.

Conclusion.

The problem of splenectomy in any particular case may be tackled by posing and answering the following questions:

Is hypersplenism present? Is hypersplenism incidental in the course of the illness, or is it the prime factor?

If it is incidental: (i) is the patient able to stand splenectomy? And if so, (ii) will the benefits of splenectomy be such as to improve the patient's chances of recovery or materially influence the progress of the disease?

If hypersplenism appears to be the main factor in the illness, the important thing is the exercise of judgement as to blood transfusion and the selection of a time for the operation.

If hæmolytic anæmia is present, is it familial or acquired? If it is familial, is there evidence of spherocytosis and of the other characteristics of familial acholuric jaundice? If so, then splenectomy at the proper time is the correct treatment.

In acquired hæmolytic anæmia, although splenectomy is not so likely to be of value, it sometimes effects considerable improvement and it may be curative.

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CORPULENCE OR OBESITY IN CHILDHOOD AND ADOLESCENCE.[1]

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If we are to discuss the subject of corpulence or obesity in childhood and adolescence adequately, we must recognize that basically it involves a consideration of the defective personality traits leading to increased intake of food and to decreased expenditure of energy.

It is of the utmost importance that we should agree to include in the programme the guidance of parents and the training of children in proper eating habits and in

[1] An abstract of this paper was read at a meeting of the Section of Pædiatrics, Australasian Medical Congress (British Medical Association), Seventh Session, Brisbane, May-June, 1950.

effective self-discipline. As pædiatricians, we must deplore the many instances we encounter of the ignorant and over-anxious mother who begs us to help her to get more food into her youngster, especially in infancy and in the pre-school years. It is our duty to lead the general practitioners in an earnest attempt to stamp out the notion, prevalent in the community, that it is necessary or even desirable to use parental pressure on the young child to eat more and more food and to drink more and more milk.

If such a crusade was successful, we would be a long way on the road to the prevention of corpulence of a disfiguring nature and the adult disasters to which it leads, such as diseases of the liver, the kidneys, and the circulatory system. Premature degenerative changes leading directly to death at an earlier age than is necessary are the consequences of those diseases after years of relative inefficiency, unhappiness and lowering of economic status.

When the distortion of the personality pattern and of the physical appearance, which we call "corpulence" or "obesity", is manifested early in life, it should be recognized, analysed and treated over a period of time long

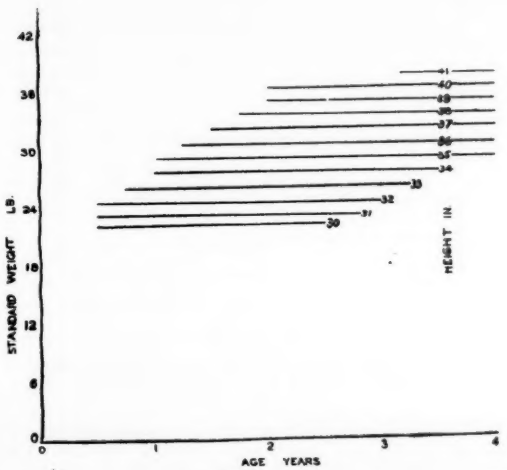

FIGURE I.
Standard weight for height at age; males, aged 0 years 0 months to 4 years 0 months.

enough to effect a correction of the defects. Half-measures are worse than useless; they count as medical failures and give support to the ideas of the parents, and of some of the patients, that the condition is irreparable. Holding those views, they feel justified in giving up their failing efforts to put up a fight against what they come to regard as inevitable.

Doctors are by no means equally able to handle the psychogenic problems which are wrapped up in the ætiology of corpulence; but the psychiatric specialists cannot yet be expected to cope with the huge volume of work which would be thrust on them if they were to treat anything like all the members of the community who are pathologically over-weight. Surely it is not necessary to call out the heavy artillery of psychotherapy (at all events often) when the patients are young. It is the true province of pædiatricians and sound general practitioners of medicine; but those who cannot exert the remedial influence should pass the patients on to others who can.

Nearly three years ago (Graham, 1947) I described a system of management which had produced many satisfactory results in my private practice. It may be helpful, at the present stage, to report again on problems encountered and progress made; certain modifications introduced as the result of widening experience will be recorded, together with epitomes of some typical case histories and some reflections engendered by careful study of the cases.

THE MEASUREMENT OF CORPULENCE.

At an early stage of the clinical investigation of corpulence it became obvious that a reliable method must be invented for assessing with precision how corpulent the subjects were at any given time. Until comparatively recently I have used the method described in 1947; but the arbitrarily chosen "size index" has been found unsatisfactory when the subject's height varies appreciably from the mean average height for sex and age. The difficulty has been overcome, and a new and satisfactory index is now in use and has been applied in retrospect to the data previously entered in the case-books.

The old system consisted of the calculation of weight index, height index and size index. The weight index is the weight of the subject, stripped or very lightly clad, divided by the mean weight for sex at age, obtained from standard tables, multiplied by 100 and expressed to one decimal place. Similarly, the height index is the height of the subject, erect and without shoes, divided by the mean height for sex at age, obtained from standard tables, multiplied by 100 and expressed to one place of decimals.

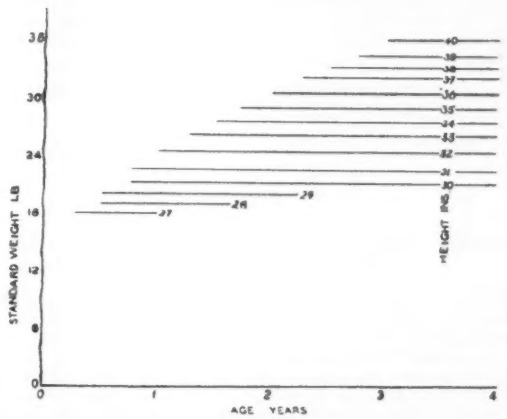

FIGURE II.
Standard weight for height at age; females, aged 0 years 0 months to 4 years 0 months.

These indices are valid and have their uses; the height index is much more reliable than the weight index as an index of growth. The size index is obtained by dividing the weight index by the height index, multiplying by 100 and expressing the result to the nearest one-half. The size index will be abandoned and the weight index will also be discarded, but the height index will be retained and the new index—the weight-height-age index—will be added.

After due allowance has been made for the type of body-build, the size or bulk of a human body is still a complex quality, of which the height is the measure of under-growth or over-growth and the other relevant component is surplus flesh, consisting mainly of fat and water, which we call "corpulence". It is already common usage to allow 13% to 15% to either side of a norm for the expected weight of a subject of broad or slender build according to the degree of divergence from the intermediate normal type of build. For many years standard weight-height-age tables have been available, and I have had extensive experience of the applicability of certain American standards to Victorian children. I rely on the Woodbury tables for boys and for girls (separately) from birth to school age and on the Baldwin-Wood tables from five to eighteen years for each sex separately; but the Australian subjects conform to the standards without the aid of clothing. The tables mentioned are obtainable from the American Child Health Association of New York City. They are not precisely suitable for the measurement of corpulence, because they are expressed in integral pounds,

inches and years and do not include data for the very tall and the very heavy boys and girls. What is wanted is a graphic method of quick and accurate determination of the standard weight for height at age by sexes. I took this mathematical problem to my nephew, Mr. Ian Graham Hodges, B.Eng.Sc., who had previously produced a nomogram for rapid (though insufficiently accurate) determination of the size index. Mr. Hodges has constructed a series of graphs, thirteen inches by eight inches in size,

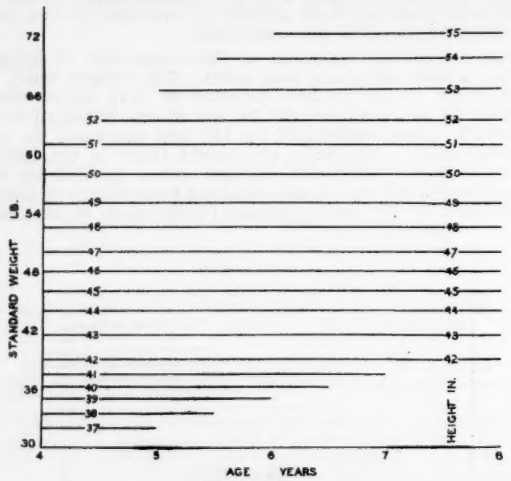

FIGURE III.

Standard weight for height at age; males, aged 4 years 0 months to 8 years 0 months.

eminently suitable for use in the office. There are five graphs for females in age-groupings of four years up to twenty years, but six were necessary for the males, two for the group twelve to sixteen years of age owing to the great dispersion of heights and weights in that age-group. It is of considerable interest to note the way in which the height lines curve through the age-weight field gracefully but with unpredictable irregularity. To use the graphs, the age to the nearest half-month is found along the base line; by moving perpendicularly upwards along a straight edge, the height of the subject is found; and by moving horizontally to the left, the standard weight is read to a fraction of a pound. The weight-height-age index (W.H.A.I.) is the weight of the subject divided by the standard weight for height at age (S.W.H.A.) multiplied by 1000 and expressed as an integer. The use of four-figure logarithms and antilogarithms is very convenient. This index is valid because it makes allowance fo the growth factor.

PROGRESS REPORT ON THE PRIVATE SERIES.

On March 1, 1950, the case-books contained detailed records of 171 children, adolescents and adults. Attainment of the age of twenty years is the dividing line in the classification of adolescents and adults. The degree of corpulence has been based on the size index at the initial consultation, but on that basis fat dwarfs are not classifiable. One hundred and forty-four subjects were under twenty years of age, 41 of them mildly corpulent, the size index being between 100 and 125; 63 were moderately corpulent, the size index being between 125 and 150; 33 were severely corpulent, the size index being over 150; and seven of them were the short corpulent individuals just mentioned. I have very carefully and rigidly scrutinized the case histories, and the assessment of results is tabulated (Table I).

The pertinent assessment groupings are the first three; they contain 103 of the cases, and only 12 are apparent failures; some have been abandoned temporarily or permanently, but some may yet be turned into successes. With increasing experience, much more convincing results have been obtained, and the subjects have achieved the objective within six months.

The Procedure, Technique and Records.

At the original consultation, identification particulars are recorded and a careful medical history is elicited from the parent or the patient, or both, including the exact date of birth, the time of onset and rate of progress of

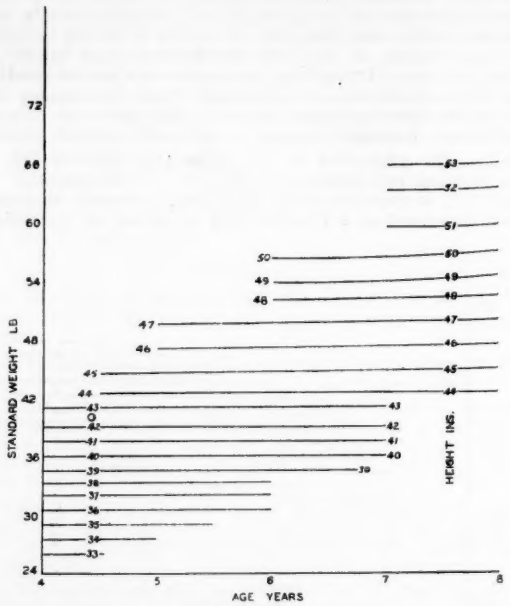

FIGURE IV.

Standard weight for height at age; females, aged 4 years 0 months to 8 years 0 months.

corpulence, previous attempts at weight-reduction, ideas about causation and reasons for seeking advice. Knowledge is gleaned discreetly about the family set-up, the eating habits and personality problems and the behaviour characteristics and interests of the patient.

TABLE I.

Assessment of Results at March 1, 1950, in 137 Cases of Corpulence.

Assessment Grouping.	Degree of Corpulence.			Total.
	Mild.	Moderate.	Severe.	
(a) Definite success ..	17	30	12	59
(b) Moderate success ..	13	11	8	32
(c) Apparent failure ..	3	5	4	12
(d) Insufficient data, short duration or gross lapses in supervision	6	12	7	25
(e) Incomplete, recent ..	2	5	2	9
Total	41	63	33	137

The patient is then examined alone in another room, stripped or very nearly stripped, and is weighed, measured and examined. Notes are made of the weight to the nearest two ounces, the height, standing erect, to one-eighth of an inch, and the age to the nearest half-month. Circumferential measurements are recorded of the chest (in full expiration, normal and full inspiration), and of the bust, when defined from the chest, the greatest girth or waist, and the greatest distance around the hips over the buttocks. The transtrochanteric diameter is also

measured at times. The type of body architecture (broad, normal, slender or mixed) and also the body mechanics (excellent, good, poor or bad) are observed, with details of posture and locomotion. Aberrations of body symmetry are noted with details about the shoulders, head, back, knees and feet. The chest is examined and the pulse rate and blood pressure (in subjects aged over eight years) are estimated. The nervous system is tested and impressions are recorded on mental, emotional and personality characteristics. A freshly passed specimen of urine is tested for albumin and for sugar, usually while the patient is dressing.

Before dressing, however, the patient is photographed alone in a room adjacent to the examination room, which is equipped for quick, roughly standardized photography. A front view, a side view and a back view are taken within three minutes (and extra "shots" as indicated), and

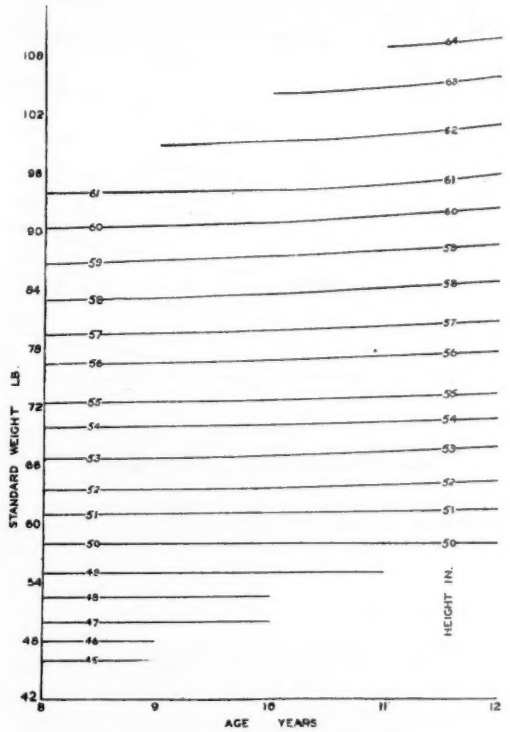

FIGURE V.
Standard weight for height at age; males, aged 8 years 0 months to 12 years 0 months.

the patient returns to the examination room to dress or be dressed. Permanent visual data are thus obtained for future use and to display the distribution and the extent of the corpulence and other features that are helpful as the case is studied.

A special record chart has been devised on which linear graphic representation of the calculated size of the patient and of the amount of thyroideum (British Pharmacopœia) grows as the case progresses; it is used to avoid empiric vagaries concerning improvement and prescription. There is provision on the chart for identification and classification particulars, details of therapy and investigations, and for general comments. A chart is started for each patient at the first interview and is placed in the case book appropriate for the age group, which is alphabetically indexed. Space is provided on the chart for weight index, height index and size index, and recently the standard weight for height at age (S.W.H.A.) and weight-height-age index (W.H.A.I.) have been added. W.H.A.I. is the index of corpulence after allowance for overgrowth; it is lower than the size index when the height is above the mean for age and is higher than the size index when the height is below that mean. The corpulence of fat dwarfs and the normality of tall children can be assessed satisfactorily by this index of corpulence; those extremes were very perplexing under the old system.

After the examination and record-making have been completed, a conference is held with the parent or the patient, or both. The problems arising are discussed and the facts are clarified, amended or amplified. Special investigations required urgently are arranged and less urgent ones may be mooted for future action. Advice is proffered on general management, including psychological aspects; general dietetic principles are stressed, including the need for reduced intake of food with proportional increase in protein and growth-promoting items and

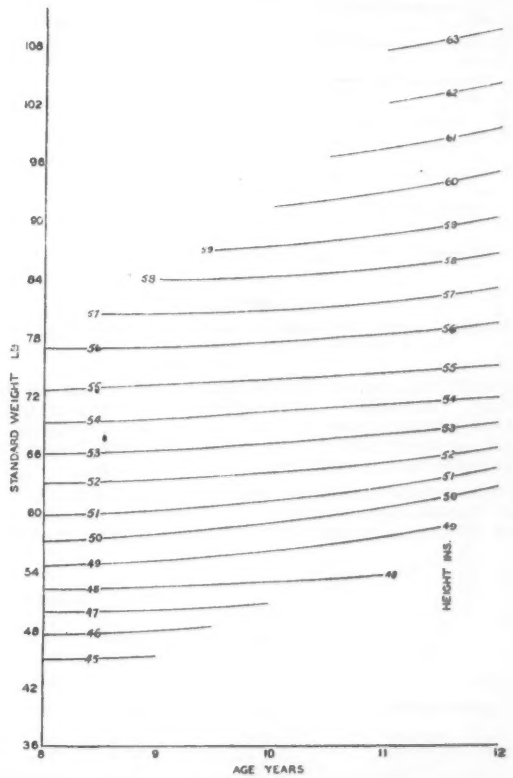

FIGURE VI.
Standard weight for height at age; females, aged 8 years 0 months to 12 years 0 months.

decrease in fats, sugars and starchy foods. Reduction in fluid intake is not emphasized, nor are detailed "diets" prescribed for children or adolescents, as they find them irksome and unnecessarily restrictive to freedom, and they are troublesome to the parents; they are bad psychologically, as they may produce in the subject a hypochondriasis or an inferiority reaction. The emphasis is placed on the development of self-control and the knowledge that will enable them to select what is good for them from the food that is offered to them anywhere. If a diet list is very earnestly desired, it may be politic to supply it. The Newburgh dietary *régime*, itemized for breakfast, dinner and tea, is the type of diet that should be specified in quantities appropriate to the size and age of the patient. No use whatever is made of purgation and no aperients have been authorized in any of the cases under consideration; even large doses of thyroideum do not produce

catharsis in these patients. Physiotherapeutic massage, baths and physical exercises are not recommended for reasons akin to those advanced above for the avoidance of "diets"; but each patient is urged repeatedly to participate wholeheartedly in sports and games. Swimming, tennis, skating and dancing are suggested to provide extra exercise with the opportunity to gain in self-esteem by proficiency. As escorts and providers of the expenses, the parents are thus given the chance to make a contribution which is appreciated by the child, and which is usually interpreted as a tangible demonstration of love.

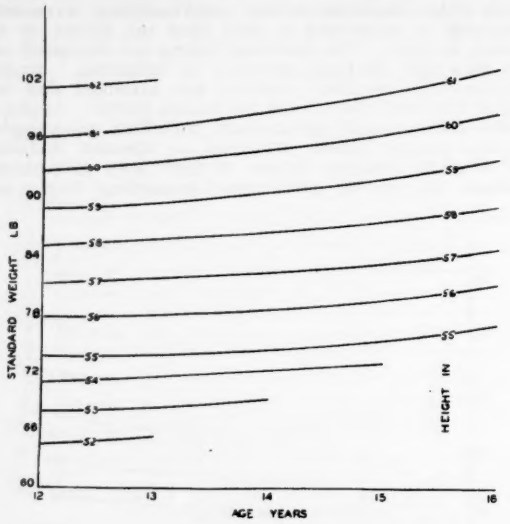

FIGURE VIIA.
Standard weight for height at age; males, aged 12 years 0 months to 16 years 0 months.

Prescriptions and specific therapy are necessary to produce results soon enough to encourage the patients and their parents to persist with the other lines of treatment advocated; the doctor also gets periodic opportunities to assess progress, to reinforce his advice and to study the problems arising from time to time. The permanence of good results depends on prolonged supervision and on the establishment of new and better habits after the eradication of the baneful ones. Chemist's supplies are likened

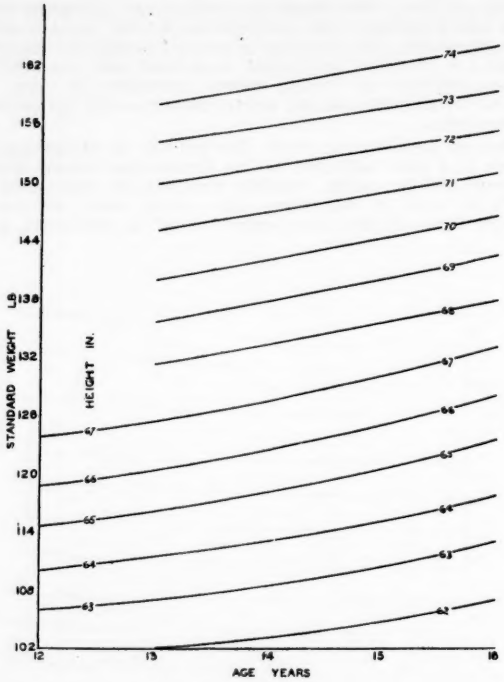

FIGURE VIIB.
Standard weight for height at age; males, aged 12 years 0 months to 16 years 0 months.

TABLE II.
Case XXIV; Female Subject; "Moderate" Series, Number 11.

Age.	Weight. (Pounds.)	Weight Index.	Height. (Inches.)	Height Index.	Thyreoid Units. (Daily Dosage.)	S.W.H.A. (Pounds.)	W.H.A.I.
11 years 10 months ..	101	123·2	55¼	96·5	2 (gr. 1½ B.P.)	76	1330
11 years 11 months[1] ..	102½	122·2	55½	96·9	4	77	1326[1]
11 years 11½ months ..	99½	118·4	55¾	97·2	5	77½	1280
12 years 1 month ..	93½	108·7	56¼	97·8	5	80½	1157[1]
12 years 2½ months ..	92½	105·9	57	98·3	5	83¼	1112
12 years 4½ months ..	93½	103·5	57⅛	98·5	6 (gr. 4½ B.P.)	85¼	1100
12 years 6 months ..	95½	104·6	57⅛	97·8	8	86½	1106
12 years 7½ months[1] ..	93½	100·0	58¼	98·3	8	88½	1059[1]
12 years 9½ months ..	96	100·0	59	99·1	8	92½	1037
12 years 11½ months ..	98½	100·0	59½	97·8	6	95	1034
13 years 0½ month ..	102	103·0	59⅛	98·5	7	96	1062
13 years 2 months ..	107½	107·2	60	98·5	8	98	1099
13 years 3½ months ..	Menarche						
13 years 4 months ..	107½	104·1	60½	98·2	7	101	1061
13 years 6 months ..	107½	101·1	61	99·0	6	103½	1039
13 years 7½ months ..	111½	104·4	61	98·2	7	103½	1080
13 years 9½ months ..	113½	103·5	61¼	98·0	7	104	1089
13 years 11 months ..	117½	105·6	61¼	97·6	8 (gr. 6 B.P.)	106	1110
14 years 0½ month ..	116½	103·9	61½	97·6	8	107½	1086
14 years 2 months ..	117½	103·3	61⅝	98·0	8	107½	1091
14 years 4 months ..	117½	103·4	61½	98·1	8	108½	1083
14 years 6 months[1] ..	124½	104·3	62	98·6	8	111½	1116[1]
14 years 7½ months ..	130	111·6	62½	98·8	10[2]	112½	1155
14 years 8½ months ..	122½	104·7	62¼	97·6	10[2]	113	1084
14 years 10 months ..	126½	107·9	62⅜	97·8	10[2]	114	1112
15 years 0 month ..	122½	102·8	62⅜	97·2	9[2]	114½	1071
15 years 2 months ..	122½	102·5	62½	98·0	8	114½	1067
15 years 4 months[1] ..	130½	108·9	62½	97·5	4[2]	115½	1129[1]

[1] Photographs available at age and W.H.A.I. shown.
[2] With two dextro-amphetamine sulphate tablets.

to crutches for temporary use during the transition period from the old state to the new, and, used as crutches to be discarded in due course, they are of the utmost value for these patients. There is a professional and lay prejudice against thyreoid medication for obesity, but I use it freely unless I find a contraindication; moreover, I have found it necessary to use relatively large dosage to achieve significant results. To cut out one confusing factor—theoretical or actual variations in potency in potentially similar products of various wholesale manufacturers—the tablets manufactured in Melbourne by the Commonwealth Serum Laboratories have been prescribed consistently. Either corpulent children have a high tolerance for oral thyreoid medication or the product mentioned is relatively impotent, which is most unlikely.

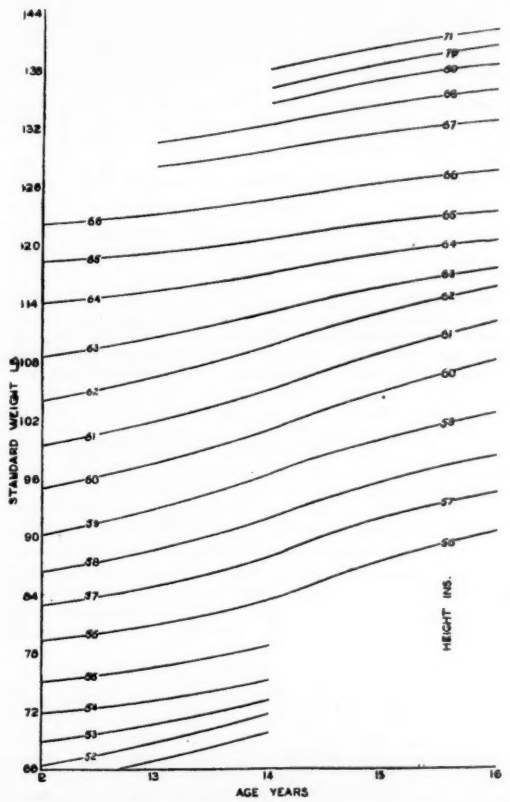

FIGURE VIII.
Standard weight for height at age; females, aged 12 years 0 months to 16 years 0 months.

The amphetamine preparations, especially dextro-amphetamine sulphate, are very useful to assist the patient to take less to eat and still experience the pleasant feeling of satiety after a meagre meal. Dextro-amphetamine sulphate has been used as a reserve weapon for older children and adolescents who are finding it difficult to gain control over the appetite; but no attempt has been made to reach maximal dosage or even to experiment with big doses. It has served its purpose on numerous occasions, but the effect wears thin as the weeks go by. It has been found best to use it intermittently for a month or six weeks now and again.

The procedure and technique thus far described usually take the best part of an hour, but no longer, and at the end of the conference cordial relations should exist and the clients should depart with a definite plan and a feeling of confidence in it. A letter of communication is sent to the referring practitioner, if reference has occurred, or to the family doctor unless this action is countermanded; but it may be advisable to examine the patient again within two to four weeks so that additional information can be collected and transmitted. It is of importance to the patient that arrangements should be made and not left to chance. It is a commonplace experience to be asked to "see the thing through" as a special project unrelated to attendance in illnesses and with the interested cooperation of the family doctor. It is an advantage, at the second consultation, to be able to demonstrate a loss of weight, however small, and an improvement in one or more of the circumferential measurements; additional emphasis can then be laid on the necessity for careful and faithful compliance with the dietary advice. Background clinical data can be completed and the details of management and

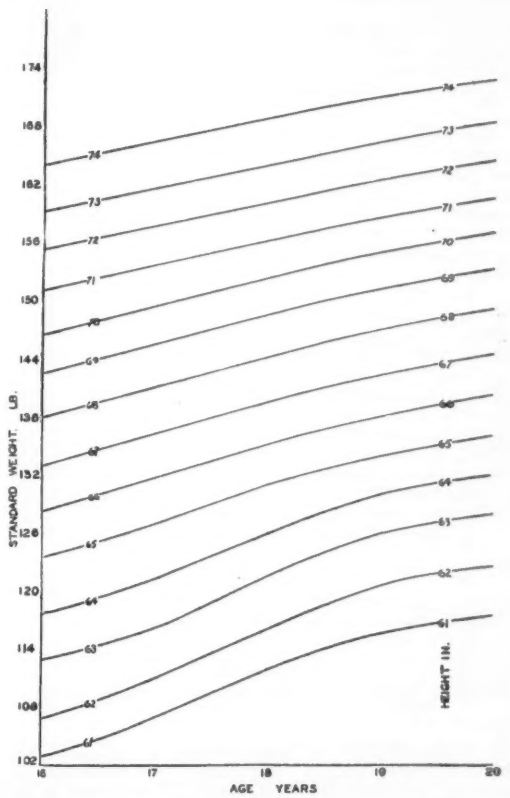

FIGURE IX.
Standard weight for height at age; males, aged 16 years 0 months to 20 years 0 months.

of treatment can be reviewed and adjusted. The second visit is thus an extension of the original consultation, and at the end of it arrangements can be made for the long-distance control of the patient. Subsequent consultations should be made once a month, approximately evenly with the birth date or to avoid menstrual periods. At each consultation the patient should be examined fairly thoroughly, with a sharp lookout for any evidence of lack of tolerance for therapy or untoward signs or symptoms. The routine measurements should be made and the calculations and progress notes completed. It is not necessary to take the blood pressure or examine the urine every time, and the photography is only occasional. It is an important safeguard to use the stethoscope and count the pulse rate every time.

Confidence, understanding and good will are built up, and no opportunity is to be lost for exhorting the patient to gain self-control and self-esteem and to hold fast to the

regimen. The child must be taught to understand what is required and the rationale of it all, and must be a willing and eager collaborator. Even very young children can be treated thus, and they appreciate the direct relationship with the doctor. As soon as material improvement has been manifested and personality control has been gained over the patient, the interval between visits should be extended to six weeks, then to two months, and later to three months; a longer interval is regarded as a lapse in supervision, but at times is unavoidable. Adjustments are made from time to time in the treatment, and when appropriate, inquiries are constantly made about the menstrual periods and about social adaptation and adjustment. Maintenance of improvement is zealously supervised for years, and any significant retrogression is an indication for more frequent visits till progress is again satisfactory. The aim is to render the patient independent of the prescribed chemist's supplies and able to rely entirely on self-control and the healthful way of life.

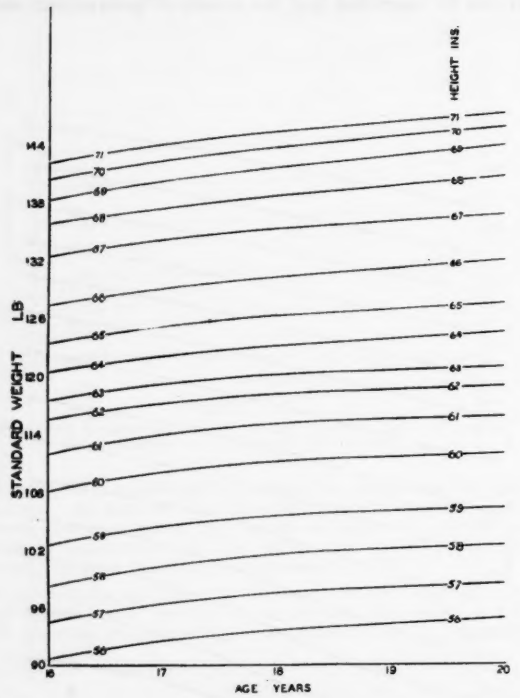

FIGURE X.

Standard weight for height at age; females, aged 16 years 0 months to 20 years 0 months.

The case summarized in Table II has been selected as an illustration because the patient has remained under constant observation for three and a half years, having been one of the few I specifically mentioned in 1947.

When the patient came under notice in 1947, at the age of eleven years and ten months, she had had left-sided hemiplegia five months earlier and was known to be epileptic. She had had an acute lesion that year also which was almost certainly upper tibial epiphysitis, and the left leg was in plaster for two months. She was over-fat (W.H.A.I., 1330) and broadly built, with lurching gait and flat feet, and she drooped at the left shoulder, but she was not clinically subthyreoid. A number of photographs are available to show varying stages in the metamorphosis that has occurred. Well within the first year of treatment the corpulence disappeared (W.H.A.I., 1037) and she had become robust, sanguine and athletic. The maintenance thyroideum (British Pharmacopœia) requirement was six grains by mouth each morning, which has been taken with nothing but benefit for over three years. The epilepsy left her at the menarche soon after her thirteenth birthday. She has become an exuberant, happy, healthy girl with a zest for life, but she still eats too much and is taking two amphetamine sulphate tablets daily in an attempt to reduce the food intake. It is to be hoped that she can soon be weaned of the thyroideum, using the amphetamine sulphate during the transition period. She is a race-winning swimmer and a bronze medallist at skating, and she dances well and has excellent school reports.

TABLE III.

Number of Age-Group Studies by Four-Year Periods and Sex.

Age Group: Four-Year Periods.	Sex.		Total.
	Male.	Female.	
Very young children (under 4 years) ..	2	7	9
Young children (between 4 and 8 years)	5	21	26
Children (between 8 and 12 years) ..	9	48	57
Pubescent children (between 12 and 16 years)	9	51	60
Adolescents (between 16 and 20 years)..	1	33	34
Total	26	160	186

TABLE IV.

Age-Group Studies Showing Degree of Corpulence.

Age Group.	Degree of Corpulence.			Total.
	Mild.	Moderate.	Severe.	
Very young children ..	3	5	1	9
Young children	7	12	7	26
Children	14	29	14	57
Pubescent children ..	15	27	18	60
Adolescents	16	12	6	34
Total	55	85	46	186

TABLE V.

Case CXLII; Female Subject; "Mild" Series, Number 35.[1]

Age.		Weight.		Weight Index.	Height. (Inches.)	Height Index.	Size Index.	S.W.H.A. (Pounds.)	W.H.A.I.
Years.	Months.	Pounds.	Ounces.						
1	10	30	13¼	121·9	33½	100·8	121	26½	1173
1	11[2]	30	0	116·5	33½	100·8	115½	26½	1143[2]
2	0[2]	31	8½	121·1	34	101·5	119	27½	1146
2	1½	31	5½	119·0	34½	101·2	117½	28	1118
2	3	31	14	117·5	36	105·8	111	30½	1045
2	4½[2]	32	2	115·7	36½	105·7	109½	31½	1003[2]
2	6½	34	0	121·4	37	105·6	115	32	1063

[1] This patient took thyreoid orally as follows: 1·5 grains daily for two months, 3·0 grains daily (B.P.) for four months and a half, and 1·5 grains daily for two months.

[2] Photographs are available at age and W.H.A.I. shown.

TABLE VI.

Case CXLVIII Female Subject; "Severe" Series, Number 29.[1]

Age.	Weight. (Pounds.)	Weight Index.	Height. (Inches.)	Height Index.	Size Index.	S.W.H.A. (Pounds.)	W.H.A.I.
7 years 1 month[2] ..	137½	266·9	53	110·9	240	65½	2091[2]
7 years 2 months[2] ..	128¼	247·8	53¼	110·9	224	66	1943[2]
7 years 3 months ..	119	229·0	53¼	111·4	207	67½	1762
7 years 4 months ..	111¾	201·3	54	112·2	180	68¼	1631
7 years 5 months ..	103	195·2	54¼	112·9	173	69½	1487
7 years 6½ months[2] ..	101½	190·0	54¼	113·4	168½	71	1426[2]
7 years 7½ months ..	98	183·1	55	113·9	161	71¾	1366
7 years 8½ months[2] ..	95½	177·2	55¼	113·9	153½	72½	1322[2]
7 years 9½ months ..	98	181·5	55¼	114·3	158	74½	1315

[1] This patient has taken thyroideum (B.P.), 7·5 grains, each morning.
[2] Photographs are available at age and W.H.A.I. shown.

Many more dramatic cases can be found in the case books of much more corpulent children who have made more satisfactory progress than the subject of this illustration; but I consider that this case serves well to demonstrate some of the difficulties and some of the gratifying features in the procedure for the conquest of corpulence in the growing child.

Clinical Studies of Corpulence by Four-Year Age Periods.

Although most of the problems of corpulence and adiposity in childhood and adolescence are of fairly general applicability, the age group to which the patient belongs poses some special points of interest. An analysis of the material in the case books has been made on this basis. One hundred and thirty-seven case histories of the patients under twenty years of age have yielded 186 age-group studies when five groups, each of four years from birth to the twentieth birthday, were formed; some of the patients passed from one age group into the next while undergoing treatment. The subjects in these age groups have been designated respectively very young children, young children, children, pubescent children and adolescents. The number of studies in each group together with the sex of the subjects is set out in Table III and the degree of corpulence by age groups is shown in Table IV.

A few of my reflections in connexion with the management and treatment are set down under the headings of the age groups; some of them are tentative and some are rather provocative. They should not be construed out of their context.

Very Young Children.

Very early childhood is the formative stage for reactions to environment. Habits, good or bad, will never be so quickly and firmly fixed at any later age; nor is it likely that the effect of the habit on the personality will ever be eradicated. The physician has only indirect control of the patient, as a rule, and that is a disadvantage. Parental instruction and guidance in the management of children must be deliberately practised, the verbiage and emphasis being suited to the intelligence and social status of the parents. The father must not be excluded and should be interviewed. Endocrinal dyscrasias, digestive disorders, mental, emotional or social inadequacies or other trends or anomalies may be found.

The heavy children are very apt to be overgrown. The height is much more dependable than the weight as a measure of growth. Close-cropped ears and extra breadth of hands and feet are the indicators of the broad type of body build. The S.W.H.A. and the W.H.A.I. are valuable data from which to assess corpulence and its amelioration; the size index, a function of comparisons of actual and mean average weight and height, is not satisfactory as a scientific index of corpulence.

It may be of interest to publish a case summary (Table V), but only those who can study the photographs will be able to judge that the final series of photographs represents relative normality as indicated by the W.H.A.I. (1003) and not mild corpulence as suggested by the size index (109⅛). In the mother's opinion the child was hard to feed and did not eat enough. Though weighing only 15 pounds at six months, she weighed 24·5 pounds at one year and 30·25 pounds at twenty months, when the height was 33 inches. The excessive weight preceded the excessive height in the first growth cycle, which can be observed in the case summary in Table V.

Young Children.

Among young children habitual over-eating looms large and is often regarded as a virtue by the *entourage*. The judgement normally guided by physiological appetite may be perverted by habitual wrong feeding in quantity and in quality; reeducation of the habits of eating and restoration of appetite to normality are often necessary.

Inherited trends to bulk of body make themselves apparent, but they must not be confused, in so far as the corpulence factor is concerned, with established though erroneous family eating customs. Inequalities in the distribution of the surplus tissues may become definable and have their effects on body mechanics. Faulty body mechanics may require orthopædic attention, and reduction

TABLE VII.

Case CXXXV; Male Subject; "Severe" Series Number 24.

Age.	Weight. (Pounds.)	Weight Index.	Height. (Inches.)	Height Index.	Size Index.	Thyreoid Units. (Daily (Dosage.)	S.W.H.A. (Pounds.)	W.H.A.I.
7 years 9½ months ..	96	178·6	53	100·2	164	6 (4½ gr. B.P.)	66½	1438
7 years 10½ months ..	91¼	167·4	53⅛	109·4	153	6	67½	1352
7 years 11½ months[1] ..	86⅜	158·4	53⅛	111·7	142	6	68¼	1267[1]
8 years 0½ month ..	84½	153·2	54¼	110·2	138	6	70½	1199
8 years 2 months ..	83½	147·3	54½	109·6	135	8	70½	1181
8 years 3 months[1] ..	79½	139·5	54½	110·3	126½	8 (6 gr. B.P.)	71½	1109[1]
8 years 4 months ..	80	140·4	55⅛	111·1	126½	8	73	1096
8 years 5½ months ..	81½	138·8	55½	110·2	126	8	73½	1106
8 years 6½ months ..	82½	140·2	55½	110·1	128	8	74¼	1111
8 years 8 months ..	85	141·3	55½	110·1	128	8	76½	1126
8 years 9 months[1] ..	84½	140·3	56¼	110·5	126	6	77	1094[1]

[1] Photographs are available at age and W.H.A.I. shown.

TABLE VIII.

Case CXLI; Male Subject; "Severe" Series Number 27.

Age.	Weight. (Pounds.)	Weight Index.	Height. (Inches.)	Height Index.	Size Index.	Thyreoid Units. (Daily Dosage.)	S.W.H.A. (Pounds.)	W.H.A.I.
10 years 7 months[1] ..	116¼	158·8	58¾	106·1	150	2	85½	1365[1]
10 years 8 months ..	112¼	151·0	58¾	106·3	142	4	86½	1301
10 years 8½ months ..	112¼	150·5	58¾	105·7	142	6 (4½ gr. B.P.)	86½	1296
10 years 9½ months ..	106½	141·5	58⅞	106·1	133½	6	87	1224
10 years 10½ months[1] ..	100¾	132·5	59¼	106·5	125	6	88¼	1135[1]
10 years 11½ months[1] ..	95¼	124·0	59½	106·4	116½	3	91¼	1043[1]
11 years 0 month ..	97½	126·6	60	107·1	118½	3	91½	1063
11 years 1 month ..	97½	125·8	60½	107·1	118	2	92	1059
11 years 1½ months ..	Tonsillectomy followed by relaxation of treatment in convalescence.							
11 years 3 months ..	108	137·6	60¾	107·5	128½	4	93½	1158
11 years 4 months ..	108½	136·9	60¾	107·2	128	6[2]	93½	1160

[1] Photographs are available at the age and W.H.A.I. shown.

[2] With one dextro-amphetamine sulphate tablet.

of weight may be sought by the orthopædist to help him to help the patient. Sudden considerable accession of weight may be symptomatic; proper investigations must be undertaken without delay. Hypogonadal phenomena may present, but specific treatment is usually futile before the patient is twelve years of age.

Bullying and cruel teasing commence to damage the personality, but the gross effects of this maladjustment may not be obvious till the child is older. Overgrowth in the first and early in the second growth cycle may cause a six-year-old to be as large as a normal ten-year-old child.

One of the grossest cases encountered has been chosen and the case summary is submitted in Table VI to represent this age group.

The father was 17 stone and the mother weighs about 15 stone and is of average height. The child is huge, with stretch-marks over the hips and breasts; the nipples are inverted with fat; she had a full-moon face, pendulant abdomen and podgy fingers. Though she was only just over seven years of age, the circumferential measurement of the hips was 39·5 inches. Treatment was commenced in July, 1949, but from the selected relevant data in the case summary and from three series of photographs the amazing improvement can be demonstrated. A thorough search has been made to discover a lesion of which the adiposity could be symptomatic, but without success.

The male of the species is also afflicted, and at this age a great deal can be done to minimize the damage to the personality. Such a one is the subject of the case summary in Table VII. The girl weighed 10·25 pounds at birth and 28·25 pounds at eighteen months, and never looked back. The boy weighed only 7·25 pounds at birth and was not regarded as outsize until four years of age; indeed, he is said to have a weak stomach and a lazy mind. Each of them is an only child, and each of their mothers is very kind and generous with the food.

The boy was known as "Fatty" at school and has mixed astigmatism and a cryptic testis. The *sella turcica* is of normal size and shape, and though he has a colossal thirst, the Benedict test result is satisfactory and the carbohydrate tolerance curve is well within the normal limits. He has made excellent physical and mental progress and has become much more active, evincing considerable interest in the games available at school. He took thyroideum (British Pharmacopœia), 4·5 grains, orally each morning for four and a half months, then 6·0 grains daily for seven months, and he is back to 4·5 grains daily as the dose for maintenance.

Precocious pubescence is indicated by outward and visible signs as well as by personality changes and forwardness of ideas and interests. One common consequence of early menarche is premature deceleration in height growth. For the stocky children the fight against corpulence is then grimmer: fat dwarfs are made thus. Delayed gonadal development is prone to be associated with obesity, and not only in the male sex. The syndrome is more florid after the age of twelve years; but the prognosis is improved with early recognition and energetic purposeful treatment aimed at the corpulence at first, and after the age of twelve years appropriate organotherapy may be used. It is advisable, in each case, to search for evidences of endocrinal organic pathological changes. The delay in gonadal

TABLE IX.

Case LXX; Female Subject; "Moderate" Series Number 29.[1]

Age.	Weight. (Pounds.)	Weight Index.	Height. (Inches.)	Height Index.	Thyreoid Units. (Daily Dosage.)	S.W.H.A. (Pounds.)	W.H.A.I.
9 years 6 months ..	96¼	148·5	55⅛	106·4	2 (gr. 1½ B.P.)	75½	1278
9 years 7 months[2] ..	96¼	145·6	55⅛	106·4	4	76½	1263[2]
9 years 8 months ..	94	141·3	56	106·6	4	77½	1217
9 years 9½ months ..	92	136·5	56¼	105·9	6	77½	1183
9 years 11 months ..	91¾	133·9	56½	106·3	7	80	1147
10 years 0½ month ..	89	128·5	57½	107·4	7	82½	1087
10 years 2½ months ..	88¼	126·1	57½	107·4	8 (gr. 6 B.P.)	83¾	1055
10 years 4½ months ..	87¾	123·0	58½	108·1	8	86½	1006
10 years 6½ months ..	91½	127·1	59½	108·9	8	89	1025
10 years 9½ months ..	95	130·1	59¾	108·9	8	90¾	1050
10 years 11 months ..	94¼	127·8	60¼	110·0	8	95	996
11 years 1 month ..	102½	137·2	60½	109·9	10 (gr. 7½ B.P.)	96½	1059
11 years 2½ months ..	103½	135·8	61¼	110·1	10	98¼	1048
11 years 4 months[2] ..	105½	136·8	61⅝	110·5	10	100½	1048[2]
11 years 5½ months ..	105½	133·9	62	110·5	10	103	1020
11 years 7 months[2] ..	109¼	140·9	62¼	111·5	10	106	1030[2]
11 years 9 months ..	111½	135·5	63	110·7	10	109½	1017
11 years 11 months ..	117⅜	141·0	63¾	111·1	10	112	1042
12 years 0½ month[2] ..	119½	141·8	63¾	111·3	10	113½	1049[2]
12 years 2 months ..	118¼	136·8	64	110·8	6	114¼	1035

[1] The menarche has not yet occurred.

[2] Photographs available at age and W.H.A.I. shown.

TABLE X.

Case IX; Female Subject; "Severe" Series Number 4.

Age.	Weight. (Pounds.)	Weight Index.	Height. (Inches.)	Height Index.	Thyroid Units. (Daily Dosage.)	S.W.H.A. (Pounds.)	W.H.A.I.
9 years 0 month ..	91½	152·1	52	101·0	2	63½	1443
9 years 3 months ..	91½	144·8	52½	101·4	3	64½	1421
9 years 6 months ..	95½	146·9	52¾	100·9	4	65¾	1452
9 years 8 months ..	91½	140·2	53½	100·4	6	67½	1347
Lapse							
10 years 8 months ..	112	151·5	56½	103·2	8 (gr. 6 B.P.)	79	1417
10 years 10 months ..	113½	152·2	56¾	103·6	10	80½	1410
11 years 1 month ..	116½	155·5	57½	103·0	10	82½	1409
11 years 2 months ..	114¾	152·1	57¼	103·5	12	83½	1370
11 years 5 months ..	120	151·5	58	103·3	16 (gr. 12 B.P.)	85½	1403
11 years 9 months ..	127	157·7	58¾	103·5	18	88½	1431
11 years 10 months ..	125½	151·6	59	103·3	20	90	1391
12 years 0 month ..	130	154·8	59½	104·1	22	91½	1421
12 years 2 months ..	124¾	143·2	59¾	104·0	18	94¼	1318
12 years 7½ months ..	Menarche. Lapse, but took *circa* 18 units in orthopædic hospital.						
13 years 0½ month ..	130¾	141·1	60½	99·6	18	98¾	1415
13 years 1 month ..	141	140·1	60¾	100·0	22	99	1424
13 years 2½ months ..	139	136·6	60¾	99·8	22	101½	1372
13 years 3½ years ..	138½	135·1	60¼	99·6	24 (gr. 18 B.P.)	101¼	1365
13 years 5 months[1] ..	141	132·9	60½	98·7	26	102	1382[1]
13 years 7 months ..	152¾	145·4	60½	98·2	24[2]	102½	1490
13 years 8 months ..	140¾	130·6	60½	97·6	24[2]	103	1366
13 years 8½ months ..	137½	127·3	60¾	96·9	24[2]	103¼	1331
14 years 1 month[1] ..	129½	115·1	60½	96·2	16[2]	104½	1239[1]

[1] Photographs available at age and W.H.A.I. shown.
[2] With three dextro-amphetamine sulphate tablets.

development may be recognized at an early age by the child's retention of the "young child" outlook, which should normally be shed as the pubescent years approach.

Over-eating is usually prominent in this age period. It may be due to greed, over-indulgence and general absence of self-control or home discipline. It may be compensatory to the feeling on the part of the child of lack of parental affection when the parent is stern, or it may be the consequence of oft-repeated depreciatory comparison of the child with a paragon, engendering self-pity and endangering rational self-appreciation. The child loses hope and no longer tries to please or to be pleasant, but gets consolation from food. Over-eating may also be due to over-abundant supplies of food and to the home habit of eating too much from sheer ignorance or misguided generosity.

Teasing and badgering by relatives and school contacts hypersensitize the child, who becomes self-conscious to such a degree that withdrawal from the herd activities occurs and may be almost absolute after the age of twelve years. Grimacing, twitching, blinking and shoulder-shrugging are apt to make their appearance, and there may be other evidences of undesirable nervous tension. The consequences are loss of healthful exercise, slothfulness, exhibitions of bad temper, retaliatory spitefulness, and in general, antisocial behaviour and failure to gain normal social experience.

The fact that this is the age of enthusiasm should be exploited by the parents, teachers and doctors. The noses of the children should be laid to the scent of happiness through health; their drooping spirits rise as the weight falls.

In Table VIII the case summary appears of a boy who was first brought along for treatment in May, 1949, at the age of ten years and seven months.

The patient was very small as a baby, and was very precious, as the parents were middle-aged and had only one other child, a boy, who was eleven years of age at that time. The five-pound baby soon became chubby and over-sized. At a leading school he was given the nickname of "Ten Ton Fat"; he developed nervous blinking and shrugging and withdrew from the games he wanted to enjoy. A complete reformation—psychological, physical and educational—occurred during treatment, marred recently by over-indulgence, relaxation of dietary restrictions, and absence of exercise during convalescence after tonsillectomy.

Another illustrative case of this age group is set out in Table IX.

The subject is a little girl for whom treatment was sought in July, 1947, because she was growing too big. The family doctor had been consulted and had been dieting her without conspicuous success; the restrictive diet was combined with the liberal administration of Epsom salt and castor oil, and she did not like the treatment. She is an only child and is precocious and super-intelligent for her years. She has been a voracious reader of the Victorian fictional classics for several years, and has cooperated excellently throughout

TABLE XI.

Case CXXIX; Female Subject; "Severe" Series Number 21.

Age.	Weight. (Pounds.)	Weight Index.	Height. (Inches.)	Height Index.	Size Index.	Thyroid Units. (Daily Dosage.)	S.W.H.A. (Pounds.)	W.H.A.I.
11 years 8½ months ..	122¾	150·6	56½	98·0	153½	10	80	1534
11 years 9½ months ..	117½	142·9	56½	98·0	146	12 (gr. 9 B.P.)	81	1447
11 years 10½ months ..	112½	135·5	56½	98·6	137½	12	82	1372
12 years 0 month[2] ..	103¾	122·3	57¼	99·6	123	10	83½	1243[1]
12 years 1½ months ..	101	121·5	58	100·9	120½	10	86½	1167
12 years 3 months ..	96	109·7	58¼	100·2	109	6	88	1091
12 years 4 months ..	97¾	109·5	59	100·6	109	7	91	1074
12 years 4½ months ..	Menarche.							
12 years 5½ months[2] ..	99	108·8	59½	100·6	108	7	92	1076[2]
12 years 7 months ..	103½	110·4	59½	100·2	110	7	94½	1095
12 years 8½ months ..	103¾	109·2	59½	100·8	108	7	96½	1077
12 years 10½ months ..	110¾	113·5	60½	99·1	114	8	102	1086

[1] Weights recorded include one to one and a half pounds for clothing; heights recorded are in stockinged feet; this affects W.H.A.I. by only 10 to 15 points.
[2] Photographs available at age and W.H.A.I. shown.

TABLE XII.

Case LIX; Female Subject; "Moderate" Series Number 60.

Age.	Weight. (Pounds.)	Weight Index.	Height. (Inches.)	Height Index.	Size Index.	Thyreoid Units. (Daily (Dosage.)	S.W.H.A. (Pounds.)	W.H.A.I.
19 years 5 months[1] ..	151½	118·4	60¾	93·4	126½	12 (gr. 9 B.P.)	114½	1323[1]
19 years 5½ months ..	147	114·8	60¾	93·4	122	10	114¼	1284
19 years 6 months ..	139½	108·8	60¾	93·4	116	10	113⅝	1216
19 years 7 months ..	134¾	105·2	60¾	93·4	113	10	114¼	1175
19 years 8¼ months[1] ..	131¼	102·1	60¾	93·4	109	10	114¾	1146[1]

[1] Photographs available at age and W.H.A.I. shown.

the time of treatment. Dermatographia has been demonstrable at times, and she is eager and excitable and was a stammerer. She has a sanguine, sunny disposition, and has become a happy, healthy, athletic and good-looking girl.

Pubescent Children.

Pubescent children, between the ages of twelve and sixteen years, comprise the largest and probably the most important group of corpulent children. This group is also the most difficult; nine of the twelve apparent failures shown in Table I belong to it. The children who came into this group via the lower age-group have been "maintained" in it with relative ease and satisfaction. Pubescence may be normal, delayed, accelerated or "abortive"; its development should be traced, when the opportunity occurs, in the progress notes; the date of the menarche should be recorded and the establishment of menstruation followed till the menstrual periods are normal. Delayed menarche leads to unusual final height growth fairly commonly, and very early establishment of menstruation is often associated with shortness and stoutness. Female as well as male examples of the adiposo-genitalis syndrome have been encountered, and the males have responded to treatment better than the females. Recognizable gonadal dyscrasias have yielded to suitable endocrinal therapy combined with the procedure described herein for coping with the tendency to adiposity.

The fight is on for independence as a free agent and for recognition as a member of the community entitled to form and to voice opinions. The physician can utilize this urge in the best interest of his patient; he must be an ally and a reliable guide and friend. Poor personal discipline, self-gratification and aggressive reactions, rather than over-eating *per se*, often account for excessive intake of food until "conversion" occurs with a change in perspective. Conversion is a psychic response to education, suggestion, release from restrictive parental mishandling and other simple psychotherapeutic measures within the ambit of most physicians. Psychiatric problems arise at times which are within the realm of the highly trained psychologist or psychiatrist.

Glowing reports from home and school have been commonplace as the patients have improved, but high achievement at skating, swimming and other sports and outstanding scholarship displayed by the patients have given me a real moral uplift.

The summaries of two difficult cases have been selected as illustrations for this age group: a more favourable case is set out in Table II. One of the subjects has been almost continuously under supervision for over five years (Table X), and the other is a recent patient (Table XI). Both the children are extraordinary characters; the former is a plausible but very mendacious extrovert, and the latter is a bad-tempered pampered introvert who is so prudish that she will not remove her clothing but is perfectly amenable to examination with her clothes on her.

The extrovert has carried a grudge against society from birth. She has severe bilateral talipes; the feet were mutilated in early operations and are misshapen and foreshortened and flattened; she has never been able to walk prettily and her gait is ungainly. She became aggressive, over-indulged and self-willed, but jolly and flamboyantly imaginative; the deterioration in her appearance with the mounting corpulence depressed her natural high spirits. The failure to curb her appetite and under-dosage of thyreoid led to a lapse in treatment for a year from the age of nine years and eight months. She returned, determined to behave better and to be lighter against the day when further operation on her feet would enable her to be as other girls. Operation was deferred several times, and ultimately her parents changed her surgeon. Meantime she indulged in flights of fancy; she became boastful about imaginary prowess at tennis and swimming, and most of her fabrications were egotistical and very plausible, though improbable. Very large amounts of thyreoid were prescribed, and though she asserted that she took all the tablets and this statement was verified by her mother, there was always a doubt of her reliability. Just after she was twelve years of age the long-awaited orthopædic operations were performed and she spent six months in hospital; by arrangement with the surgeon she was given the thyreoid prescribed, but without material benefit. Amphetamine sulphate was added recently, and at long last significant reduction has occurred. She has become gay again, and the future is brighter.

The introvert child, the subject of the case summarized in Table XI, was at first a puny weakling, but she became progressively more corpulent after seven years of age. Being petted and over-indulged, she became capricious, prudish, obstinate, rebellious, resentful and generally sour-tempered and over-emotional, with a poor scholastic record. The parents are dour and stern, and she had no doubt that they were very disappointed with her. The real personality is emerging during treatment which is in progress; already the transformation has amazed her friends and relatives, but there is still a long way to go before she can be considered to be normal.

Adolescents.

A surprising number of adolescents and young adults have consulted a pædiatrician for the treatment of corpulence; many more must want this attention from other types of practitioner. Varying degrees of maturity of intellect and personality are met in the adolescent age group, but nearly all the clients want to be regarded as adult, and this foible should be utilized by the physician for the benefit of the patient; he can at least keep the conversation at the adult level and agree only grudgingly that there is room for improvement in the lady's appearance.

Those with corpulence of mild degree are recorded as "cosmetic". After leaving school and entering the social sphere and the competition for employment and for "boy friends", some girls become self-conscious of real or imaginary corpulence to the point of seeking medical aid. Others know that they look rather attractive, but feel that their physical charm would be enhanced by weight reduction. The cumulative effects of excessive growth from endogenous causes are productive of the "large-framed" person; due allowance must be made for this growth factor in calculations of the corpulence factor.

Many of the adolescents in the group have graduated from the lower age-groups, and it may be pertinent for the pædiatricians to ask themselves at what stage their patients are no longer children. When one has had the privilege of being a valued influence in a child's life, it seems desirable to go on until the job is finished and maturity is reached.

Some of the adolescents are what they are through want of self-control earlier in life, and those are unpromising

material. The breaking of bad habits of long standing requires the skill and technique of the psychiatrists; the patients have often flitted from one doctor to another and will not heed any of them, but continue to go their own foolish way. It is not surprising that they have to pay the price and stay corpulent.

The case summary chosen to illustrate the group of adolescents (Table XII) is somewhat spectacular because of the speed with which a good result was obtained—which may or may not be maintained.

The subject of it is a stenographer who was dumpy and big-breasted, but relied on merriment rather than cosmetic aids to beauty. The mother and her family are solidly built and broad and the father and his family are relatively slim. The girl has a twin brother who is slightly under average height and weighs about twelve stone. Her birth weight was only three pounds, and she was fairy-like and quite slender till a few months before the menarche at thirteen years and six months. She has always been strong and healthy, and had no interest in boys as she grew fatter and fatter in the past year or two. She moves actively and jerkily, but considered she did not have enough exercise; she is fond of riding horses and her bicycle and has the chance to do so at the week-ends, but that does not satisfy her. She is delighted with the progress she has made in a few months, and has improved her appearance with cosmetics, smart dresses, high-heeled shoes and more becoming arrangement of her hair. Her height varies from 60⅜ to 60½ inches, depending on the effort she makes to stand erect; 60¾ inches has been used in the calculations.

SUMMARY.

1. In a previous paper (Graham, 1947) a discussion was presented of constitutional and ætiological aspects of corpulence and the rationale of psychotherapy, dietetic control, physical activities and adequate oral thyreoid therapy as a regimen for its amelioration. Mensuration and record-keeping were described and a nomogram for ready-reckoning was provided.

2. The present paper is a progress report on the private clinical research project, and as repetition has been avoided it should be regarded as a continuation of the previous paper.

3. An improved system of assessment of corpulence is described, involving the use of specially constructed charts for rapid estimation of standard weight for height at age which, when compared with actual weight at that height and age, provides the basis for a purer index of corpulence called the weight-height-age index.

4. Emphasis is placed on psychotherapeutic measures in considerable detail and in relation to the special problems of each four-year age group from birth to twenty years of age.

5. The role of thyroideum, amphetamines, gonadotrophic medications and other drug store supplies is likened to the temporary, though greatly appreciated, use of crutches; the objective is to reach the happy stage when they can be discarded.

6. Certain indications are mooted of a preventive nature, and the need for thorough treatment by competent doctors and for prolonged supervision to prevent recidivation is supported by argument and clinical evidence.

7. Personal procedure, method and record-keeping, based on widening experience, are described in detail, to be an example from which others who can may build techniques for providing a more efficient medical service for those who need it. Over-weight is far too common; and the unchecked sequelæ amount to a major human catastrophe which is a reproach to us all.

REFERENCE.

Graham, H. Boyd (1947), "Corpulence in Childhood and Adolescence: A Clinical Study", THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, page 649.

Reports of Cases.

DIVERTICULUM OF THE THIRD PART OF THE DUODENUM.

By RICHARD FLYNN AND H. HARRIS,
Sydney.

E.P., a female patient, aged fifty years, sought medical attention on December 6, 1949, because of pain in the abdomen, poor appetite and loss of weight. She had been well until the previous July, when she had noticed epigastric distress. The distress occurred within half an hour after meals and usually lasted till she gained relief by ingestion of alkaline powder. The pain occurred daily and even woke her at night. She had lost ten pounds in weight and her appetite had been poor. Since the onset of the pain she had experienced hot and cold flushes when the pain was present. She had had two previous operations elsewhere, one for the removal of her appendix and the other a pelvic exploration.

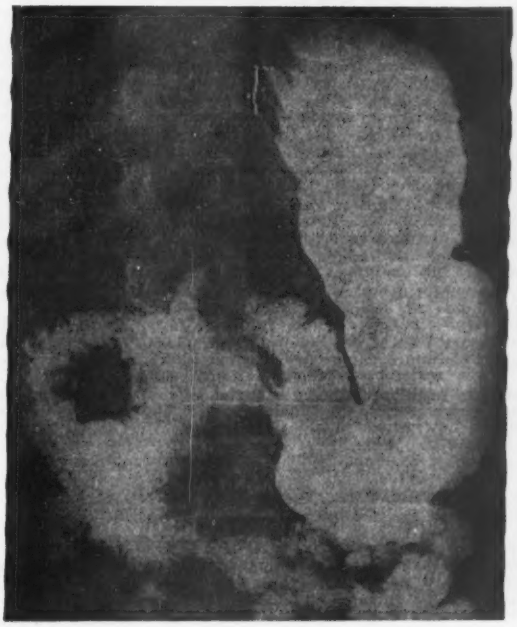

FIGURE I.

Physical examination revealed an area of hyperæsthesia in the epigastric region, with much tenderness and muscle guarding. A test meal examination revealed moderate hyperchlorhydria and also a trace of blood. Dr. K. J. Cronin reported on a barium meal examination as follows: "No ulcer in the stomach or duodenum, but there is a well defined diverticulum coming from the duodenum close to its junction with the jejunum. Evacuation is complete in one and a half hours." (See Figure I.)

After consultation with Dr. C. G. McDonald, it was decided that the diverticulum was the cause of her distress, and operation for its removal was advised.

Operation was performed on January 5, 1950, and the diverticulum was isolated and excised. The patient has been entirely relieved of pain since that date. She returned on February 6 for post-operative X-ray examination. Dr. G. C. Potts reported as follows: "No abnormality was seen in stomach, œsophagus or duodenum. The duodenal loop was regular and showed no scarring from the operative interference. The diverticulum has been removed."

The case is reported because diverticulum of the third part of the duodenum is an unusual cause of upper abdominal pain.

Reviews.

PRINCIPLES OF PHYSIOLOGY.

IN the preface to the tenth edition of his well-known book "Principles of Human Physiology" (originally the work of E. H. Starling) C. Lovatt Evans states that "for the ultimate benefit of reviewers" he will not list the main features of the new edition, but will "leave the matter to their discernment".[1] In actual fact not a great quantity of new material has been added, but much rearrangement has taken place. Some of this rearrangement is dictated by the insertion of new material or the deletion of old; some is designed to improve on the previous edition; some seems to be just "for the ultimate benefit of reviewers".

The difficulty of the author of a standard work such as this may be appreciated with sympathy in view of the spate of literature that has cascaded from the medical Press since the end of the second World War. It is a problem to be aware of it all, let alone to decide which of it is destined for the main stream of knowledge. Professor Evans has, probably rightly, been cautious. He has inserted brief accounts of a good deal of recent work, with references to important papers and books, but has made few lengthy additions; as a result he has added only 39 pages to the book without discarding much previous material. Sixty new figures have been added or substituted for old, the over-all total being increased by 25. Subjects on which new material is inserted include heat formation and free energy, the energy rich phosphate bond, chromatography, X-ray measurements of the eye, hypotheses of normal refraction, fainting, *nor*-adrenaline, growth (in relation to food intake), vitamins, the metabolism of tissue slices, the intermediary metabolism of carbohydrates, enzymes, colour vision and the Rh factor. Chapter VIII has been rewritten under the heading "Bioelectric Potentials", some of the material on electrical changes in living tissues having been transferred to the previous chapter. A number of sections have been modified, for example, that on the localization of function in the cerebral cortex. In general, considerable effort has been made at least to touch on significant changes in physiology; perhaps some subjects, for example, the Rh factor, are treated too briefly. The method of revision used tends to produce a patchwork effect and cannot be expected to prove satisfactory for too many editions. This is a common fault of standard text-books, the re-writing of which is a formidable undertaking; but new wine does demand new wineskins.

THE TREATMENT OF BURNS AND SCALDS.

LEONARD COLEBROOK is internationally famous for his researches on the bacteriological aspects of burns and scalds. Consequently, his monograph, "A New Approach to the Treatment of Burns and Scalds",[2] an epitome of his work in the Burns Unit at the Birmingham Accident Hospital, merits the closest attention.

Colebrook makes a plea for the establishment of specialized burns units, explaining how the Burns Unit of Birmingham has fully justified its existence, firstly by the reduction in mortality and morbidity of its patients, and secondly in the provision of increased opportunities for research into all aspects of the problems of burned and scalded patients.

After some rather harsh but, to a certain extent, true criticism of the treatment of the badly burned patient in the general surgical wards of a general hospital, the work of the burns unit is carefully detailed. Colebrook points out that after immediate and modern resuscitation has saved extensively burned patients, who a few years ago would have surely perished, prognosis then depends on the control of infection. Therefore, particular attention is paid to this control in all phases of treatment. This is done at Birmingham by nursing patients in separate cubicles and by the use of a strict method of dressing the burned areas. The careful methods of the operating theatre are used, and each patient is dressed in a special dressing room. Here the room air is constantly changed by a high-efficiency Plenum system of reasonable simplicity. All these methods which aim at the prevention of infection and which therefore allow early healing or grafting where necessary, are under the supervision of a bacteriologist who is designated the "Infection Control Officer".

The results of the burns unit are so imposing that it would be well for hospital planners and administrators to devote a great deal of thought to the establishment of similar units. A suggested layout of such a unit attached to a general hospital is appended with suggestions about staffing and running. It has been said that there would not be enough patients for such a unit in Australia. However, Colebrook states that it was not until his burns unit was formed, for the concentration of patients who would otherwise have been scattered in many hospitals, that the magnitude of the number of burned and scalded patients in Birmingham was realized. In that city, with a population of a little over a million, there are each year 500 in-patients and 2000 to 3000 out-patients with burns or scalds. At least 60% of these are now treated in the burns unit.

It is interesting to note from the public health point of view that here, as probably elsewhere, 60% of the burns and scalds occur, not in the factory, but in the home, and are due to what Colebrook describes as three of Britain's national habits: the employment of open fires for heating; the widespread use of highly inflammable cotton materials with a "raised" nap (flannelette) for garments for women and children; and the frequent drinking of tea. Of these home burns 55% and of the scalds 44·5% could have been prevented by reasonable adult care and foresight. Colebrook advocates the adoption of public health measures to eliminate the three national habits mentioned by him. He mentions, too, that burns units can be used to train the personnel who may be required in the event of further warfare in which burns are likely to be the cause of many casualties.

This book is thus of great interest to a wide circle of readers. The general practitioner, the surgeon, including the plastic surgeon, the bacteriologist, the hospital planner and the public health medical officer will all find something of interest in its pages.

X-RAY DIAGNOSIS.

A RATIONAL BOOK of modest size comprising the elements of modern radio-diagnosis has long been needed. Such a book is "X-Ray Diagnosis" by G. Simon.[1] Its reading by all senior medical undergraduates in this country would do much to fill the radiological hiatus existing in our present medical curriculum, and there can be no question that every hospital resident and general practitioner should know at least as much of the science of radio-diagnosis as is presented in its two hundred odd pages.

The author's aim is primarily to build an understanding of the indications for the use of X rays in diagnosis; and, without delving into the intricacies of differential diagnosis, he indicates those diseases in which pathognomonic X-ray signs occur and those in which the results need to be correlated with clinical and other findings. This is calculated to help the learner to acquire a true sense of values in his use of X rays and in his application of a radiologist's report. The radiological appearances are concisely and accurately described.

An introductory section dealing with the technical aspects of the subject is ample to allow an understanding of the types of apparatus used and of the various forms of investigation available.

The remainder of the book is classified on a useful regional basis and the scope is complete. The author's sound common sense and simplicity of style make easy reading and the numerous illustrations are well chosen. The important subject of timing of the X-ray examination in the course of an illness is always dealt with adequately. Useful notes on such diverse procedures as tomography, angiocardiography, mass radiography of the chest *et cetera* are included. If any criticism can be made it is only that some statements on controversial questions are rather too dogmatic. Such an approach, however, is doubtless intentional and makes for a lucid and concise presentation. The general standard of the publication is high and the numerous reproduced radiographs are of good quality.

[1] "Principles of Human Physiology" (originally "Starling's Principles of Human Physiology"), by C. Lovatt Evans, D.Sc., F.R.C.P., F.R.S., LL.D. (Birmingham), with a section on the special senses by H. Hartridge, M.A., M.D., Sc.D., F.R.S.; Tenth Edition; 1949. London: J. and A. Churchill, Limited. 9¼" × 5¾", pp. 1210, with 693 illustrations. Price: 42s.

[2] "A New Approach to the Treatment of Burns and Scalds", by Leonard Colebrook, F.R.S., F.R.C.O.G.; 1950. London: Fine Technical Publications. 8½" × 6", pp. 174, with illustrations. Price: 12s. 6d.

[1] "X-Ray Diagnosis for Clinical Students and Practitioners", by G. Simon, M.D., B.Ch. (Cantab.), F.F.R.; 1949. Cambridge: W. Heffer and Sons, Limited. 9½" × 7½", pp. 220, with 179 illustrations. Price: 20s.

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THOUGHTS ON AN OBSOLETE DRUG.

IN the history of medicine some of the most revealing backward glances are those which recall the pharmacopœia of a vanished day. With the newest antibiotics on the modern pharmacist's shelves, ready for the asking, we can afford to look with some scorn on the outdated ritual of the imposing coloured glass jars in the windows of his predecessor, who wrote "Dies Saturni" in his day-book and dispensed those galenicals in which our fathers rested their faith. Seventy years ago an *Extractum Liquidum* and a *Succus Conii* were official preparations of the British Pharmacopœia. Fifty years ago conium and its eight preparations and even the alkaloid coniine and its salts were pronounced as no longer official, and given that half-farewell which consigns drugs to eventual oblivion. When we look back to 399 B.C., and recall the fatal cup of hemlock that set free from the robust body of Socrates his still more robust spirit, we may wonder if it was the same *Succus Conii* which in some ætiolated form held its place till comparatively recent years in our pharmacopœia.

Perhaps it is not without interest to speculate on the method of preparation of this nauseous compound which shook neither the physical nor spiritual calm of the greatest citizen of Athens. When conium was used in medicine the concentration of the active principle was known to be very variable. Did Socrates in his wisdom have some doubt of the efficacy of the fatal draught? When the servant of the magistrates, unmanned by the philosopher's serenity and fortitude, burst into tears, Socrates asked that he whose duty it was should bear him the cup, or failing him, he who was entrusted with the task of bruising the hemlock. Calmly the sage emptied the vessel, and following instructions given him, walked about until weakness in his limbs warned him that the cold hand was upon him, and then lay down. Still able to speak, he warned his friends that the poison which chilled his limbs was mounting to his heart, and so, with one last quiver, the sturdy body gave up its spirit. Medically speaking, we assume that the dose of the drug was ample, ensuring muscular paralysis, without convulsive effect, and at last causing respiratory paralysis. So passed that noble soul; perhaps he was as he himself said, "the gadfly of Athens", yet one who, despite the injustice of his indictment, taught young and old, poor and rich, wise and foolish, to think for themselves. As Grote has said, no other philosophers "either produced in others the pains of intellectual pregnancy, or extracted from others the fresh and unborrowed offspring of a really parturient mind".

By what ironical chance did Socrates linger in prison for some weeks before his death, there to pursue the consideration of immortality with his friends? It was indeed curious that the day before his trial the sacred embassy of a ship was sent to Delos, and until it returned no one could be put to death by public decree. This ship commemorated the saving of the twice seven young children who were carried to Crete by Theseus, before he slew the Minotaur, whose victims they were to be. So, on the day before the trial of Socrates, on a charge of changing old gods for new, and of corrupting the young, a festival began to celebrate the saving of the young, and when through impeding winds and seas the ship at last returned, the philosopher was released, first from the iron bondage of his chains, and then from the bondage of the flesh.

We cannot here follow the elevated subtlety of the dialogue known as "The Phædo"; we can only note that it combines close and consistent reasoning with an ever-present sense of the vision of the world unknowable and inapprehensible of that mystic of a much later day, Francis Thompson. Without violating good taste we may even reflect how strange it is that a drug which once gained such notoriety, and centuries afterwards won a semi-scientific place as a sedative and antispasmodic, should now be destined to be forgotten. Hemlock no longer interests us botanically or pharmacologically. Indeed the name itself is confusing to those who do not know that in North America the hemlock is a variety of spruce. We may recall Longfellow's reference to "the murmuring pines and the hemlock", and Whitman's praise of the outdoor pleasure of "the bed of hemlock boughs and the bearskin". Turning again to the humble wayside member of the Umbelliferæ, we do not know if the hemlock of the Athenians was the common maculate variety, or the water hemlock, and now it does not matter.

So will pass many a drug once famous. Perhaps some analytical chemist will pause awhile to unravel the molecular structure of a lowly alkaloid or glucoside, and then rebuild by brilliant synthesis some bold new drug, potent, and maybe, in cautious modern term, relatively non-toxic. Perhaps some mycologist will investigate a still more humble mycelial tangle, and by feeding it on an appropriate diet, isolate still another antibiotic, which will vanquish rickettsiæ and viruses almost before the laboratory can prove their presence. Today is not the day for astute empirics like Paracelsus; even an inspired guess will not carry us very far. More than ever we need to know details of the cellular metabolism of the organs whose disturbances we try to correct, and even the enzyme systems of the bacteria which attack them. Obviously the mind of a practising doctor cannot encompass these fields, yet we hope never to see the day when the "push-button era" of the popular Press arrives in the practice of medicine. In our student days the Socratic

method of question and cross-examination was used by our teachers, sometimes to our discomfort, but always to our good. The teaching generation of today can use no better triad of subject, method and doctrine than those employed by Socrates. His subject was human affairs, his method, accuracy in definition and classification, and his doctrine his theory of virtue, with its constant regard for the consequences of actions. Unfortunately the publicity given today to illness and the newest and most active remedies tends to obscure the nature of health. Again we may look to Socrates, who pointed out that health and strength were not in their essence things material which could be touched and handled. In fact he taught that "we can never truly and in reality acquire wisdom through the body". These truths were too hard for Athens of twenty-five centuries ago; let us hope they are more assimilable today. We have been warned by eminent Australians against complacency. In the medical world we have the corrective of the foundation sciences, and the national university takes a forward step in laying emphasis on these as the basis of medical research. It will be for the well-armed practitioner of tomorrow to keep that self-discipline which is the hall-mark of cultivated men. Patients make up the public, and for guidance in matters of health they look to those advisers who live and move among them day by day.

Current Comment.

THE TREATMENT OF CŒLIAC DISEASE.

The dietary treatment of cœliac disease based essentially on bananas is well known and approved by pædiatricians. It was first introduced by Sidney V. Haas in a paper read before the Section of Pediatrics of the New York Academy of Medicine in November, 1923, and published in 1924.[1] Its use has brought much success in the management of cœliac disease, though there have been failures, and it is of great interest to read a recent paper by Haas, written in conjunction with Merrill P. Haas,[2] in which is summarized twenty-five years of experience since the significant dietary management was introduced. In his original paper Haas described cœliac disease as "a nutritional disturbance of late infancy and early childhood, due to inability to utilize fats and carbohydrates in a normal manner". He reported successful results from administration of a diet of high caloric content containing unrestricted protein, ripe bananas and a lactic acid milk preparation; no other form of carbohydrate or fat was permitted. Since then he has completely modified his views on fat utilization, and the recent paper describes cœliac disease as "a protracted, intermittent diarrhœa of children caused by the ingestion of carbohydrates other than those found in fruits and to a lesser extent in vegetables and in protein milk". The specific carbohydrate diet which has been found to cure cœliac disease is based on the use of banana and protein milk, properly prepared, with the addition later of other fruits and some vegetables, *plus* meats, fish, fowl, eggs, gelatine, and natural cheese. Fats are given in normal amounts. Haas and Haas state that symptoms disappear and nutrition progresses if this diet is followed strictly and forbidden carbohydrates are excluded; relapse may follow the ingestion of forbidden carbohydrates even in small amounts, such as a single lollipop or a teaspoonful of ice cream. Cure results if the treatment is maintained for a sufficient time, usually eighteen months. Haas and Haas report that they have examined 603 children who were diagnosed as having cœliac disease, but only 370 have been adequately followed. Of these 370, 357 have been cured or are on the road to cure; the standard of cure is toleration of a full normal diet at the end of an arbitrarily established period of three years. Eight children were not cured; of these, five neglected the diet requirements, one was cured after four and a half years and one after six years, and one suffered relapse after asthma. Three children died from intercurrent infection. These are excellent results, which others may have failed to reproduce; but one of the important features of the paper is the thorough and detailed discussion of the diet, and it may be that those who have had more failures than Haas and Haas will find an answer to their difficulties in this paper. The extensive experience of the authors suggests that at least their detailed views on diet warrant careful trial. Another interesting feature of the paper is a discussion of conclusions about the nature of cœliac disease and its features that might be drawn from the empirical findings in the management of the disease. Several hypotheses are put forward, quite tentatively; though we shall not attempt to go into them here, they are commended to those interested in solving the mystery of this disease, the ætiology and pathology of which are very little understood.

[1] *American Journal of Diseases of Children*, October, 1924.
[2] *Postgraduate Medicine*, April, 1950.

VAGOTOMY AND PEPTIC ULCER.

On the last occasion on which reference was made in these columns to vagotomy in the treatment of peptic ulcer (January 21, 1950) we quoted, amongst others, a physician, T. Grier Miller, who in an editorial article in *The American Journal of Medicine* had indicated an attitude of considerable reserve, and of partial disfavour, towards the operation. He went so far as to suggest the temporary abandonment of the ready employment of vagotomy until a longer period of evaluation of results was available. As might have been expected, his statements have now been taken up by a leading advocate and pioneer of vagotomy, Lester R. Dragstedt, whose reply appears in the editorial columns of the same journal.[1] Dragstedt states that since complete vagotomy as a method of treatment for peptic ulcer was introduced in January, 1943, 580 patients have been operated upon by this method in his clinic. "The procedure has gradually made its way until now it has supplanted subtotal gastric resection for duodenal and gastrojejunal ulcer on the five surgical services in the Albert Merritt Billings Hospital. Dr. Walter L. Palmer and his associates of the Department of Medicine likewise request vagotomy and gastroenterostomy in preference to subtotal gastrectomy for patients referred for surgical therapy." That is so much for the positive side. On the defensive side, Dragstedt expresses regret that Miller has called attention to the reports of those clinics only where the results of vagotomy have been more or less unsatisfactory and has not referred to Dragstedt's clinic or the Cleveland Clinic, which have the largest series of cases and results more favourable than those secured by subtotal gastric resection. Replying to specific points, Dragstedt states that some of the objections to vagotomy raised by Miller depend upon reports that he (Dragstedt) does not believe to be correct; for example, that complete section of the vagus nerves is incompatible with life. Both his experimental work and his operative experience conflict with this last-mentioned statement; the outstanding illustration is the excision of a large section of the vagus nerves during the resection of the lower part of the œsophagus for benign stricture or malignant disease. "The improved nutrition that has resulted in those patients with carcinoma of the lower œsophagus who have survived resection is ample evidence that, in man, complete vagotomy does not produce a fatal failure of digestion or absorption, or in the function of any of the abdominal viscera." The reduction in pancreatic secretion that has been reported is not of practical importance in view of the high degree of pancreatic reserve that has been shown both clinically and experi-

[1] *The American Journal of Medicine*, April, 1950.

mentally after various degrees of pancreatic resection. Dragstedt refutes Miller's suggestion that the relief of pain almost always reported after vagotomy for duodenal ulcer is presumably due to a lack of tonicity of the gastric wall; he has reproduced the typical ulcer pain in all its severity by instilling dilute hydrochloric acid into the stomach during the first five to ten days after vagotomy, the period when the depression in gastric motility is greatest. This observation also indicates that vagotomy does not anæsthetize the stomach and so produce a danger of painless recurrence of ulcer or painless perforation. In reply to Miller's suggestion that vagotomy alone is contra-indicated in gastric ulcer because of the difficulty of ruling out a malignant lesion, Dragstedt agrees for lesions in the lower half of the stomach. However, he states, in partial gastrectomy for ulcer in the upper half of the stomach the line of transection comes so close to the lesion that nothing is accomplished in the way of therapy should subsequent examination establish the presence of carcinoma. Total gastrectomy offers the only prospect of cure for carcinoma in this area, but is not considered justified with its risks and disadvantages in the absence of a positive diagnosis of malignant change. Dragstedt has seen vagotomy and gastro-enterostomy produce rapid healing of a number of these juxta-œsophageal gastric ulcers in which malignant change could not be demonstrated. So Dragstedt returns the ball to the critic's court. He admits the shortness of the present period of observation and the technical difficulties of operation and post-operative management; but he is convinced of the ætiological soundness of the procedure and looks to further experience to produce better clinical results. It is obvious that in at least one surgical clinic there will be no temporary abandonment of the ready employment of vagotomy.

"MEDICAL SYMPATHECTOMY" AND HYPERTENSION.

Sympathectomy has now had quite extended use in the treatment of hypertension, but no final verdict can be given on its value. Its results are variable and unpredictable, sometimes pleasing, symptomatically at least, sometimes disappointing. Some clinicians remain to be convinced of the operation's usefulness, some are opposed to it, some favour it. Most will concede that the theoretical basis of the operation is not clear, and many will agree with Richard Turner[1] that "sympathectomy is an unscientific method of dealing with hypertension". Equally unscientific, Turner admits, is "medical sympathectomy", "but it is a gentler procedure and would be preferable if equally effective". This production by drugs of effects comparable with those of surgical sympathectomy—"medical" or "chemical sympathectomy"—is a more recent development than the surgical procedure, but it cannot be said that its clinical value and theoretical basis are any clearer. Mark Nickerson[2] puts the position thus:

> . . . any assessment of the place which these agents may ultimately occupy in the therapy of hypertension depends upon a more specific delineation of the role of the sympatho-adrenal system in human hypertension. In spite of extensive laboratory and clinical investigation and elaborate speculation this role is still obscure.

He proceeds to discuss this role in the light of present knowledge and lists three groups of "adrenergic blocking agents". These he defines as "agents which block responses of effector cells to sympatho-adrenal stimuli". Chemical blockade, he states, may occur at many points along the reflex arcs controlling the activity of the sympatho-adrenal system; but to achieve a desirable degree of specificity the blockade must be produced at the efferent neuro-effector junction. The characteristics of an adrenergic blocking agent capable of producing useful "chemical sympathectomy" are high specificity, ability to produce a blockade effective against strong stimuli, a prolonged and uniform action and (especially important) a high therapeutic index. Of the compounds currently employed for the production of adrenergic blockade, the β-haloalkylamines appear to be the most promising. Nickerson deals in some detail with "Dibenamine", which "may be considered the prototype" of the group, and describes the members of the group as the most specific, effective and persistent of the available agents. The second group belongs to the ergot alkaloid series. The dihydro ergot alkaloids appear to be much more effective than their non-hydrogenated congeners. However, at present much of the experimental work on these compounds is complicated by the severe depressant effects that they exert upon vasomotor reflexes and the vasomotor centre and by their stimulant action on the vagal nuclei. The clinical administration of the dihydro compounds has been seriously limited by their powerful emetic action, and significant specific adrenergic blockade has not yet been produced in humans by members of the ergot series. The members of a third group of adrenergic blocking agents that has received recent attention, the imidazolines ("Priscoline" is the most thoroughly studied of them), appear to cause so much cardiac stimulation that no consistent lowering of the blood pressure is observed.

The effect of satisfactory adrenergic blocking agents is to lower the blood pressure in cases of neurogenic (sympatho-adrenal) hypertension. Nickerson points out that at present pheochromocytoma and intracranial lesions are the only causes of human hypertension that are known to involve overactivity of the sympatho-adrenal system. However, he states that presumptive evidence is accumulating to indicate that neurogenic factors may be involved in early essential hypertension, and it is possible that adequate adrenergic blockade early in the course of such hypertension may be effective in aborting it. Only additional evidence on the ætiology of essential hypertension and further clinical trial of adrenergic blockade can make this clear.

The possible value of methonium compounds in producing "medical sympathectomy" for hypertension is the subject of two British reports.[1] They are both essentially clinical in character. In the first of these Richard Turner, whom we have already quoted, presents a preliminary report based on six months' experience. He points out that the desirability or otherwise of reducing the blood pressure of patients with severe hypertension is a question on its own and one that is by no means settled. A high pressure is probably but one manifestation of hypertensive disease, and not necessarily the most important; but so long as the pathogenesis remains unknown, all treatment must be empirical, and at present it is mainly directed at reducing the pressure. This effect is produced by pentamethonium and hexamethonium compounds. Turner states that, strictly speaking, the term "medical sympathectomy" does not correctly describe the action of these methonium compounds, which is not confined to one part of the autonomic nervous system, but for practical purposes this is their main effect. Turner describes his experience with the intravenous, intramuscular and oral administration of the iodide and bromide of hexamethonium and pentamethonium and the practical difficulties (mostly not insuperable) associated with their side-effects. He believes that they have as yet no place in the routine management of patients, though they may prove useful in particular cases in the treatment of resistant symptoms related to hypertension. In the second of the two articles Stephanie Saville reports the results obtained with pentamethonium iodide in the treatment of five patients suffering from progressive hypertension with severe symptoms. In all cases it relieved troublesome symptoms and appears to have arrested the progress of the condition—for how long cannot yet be said. In this may lie its practical value, even though the systolic blood pressure is not always kept very much lower than it was before treatment. The reduction of an abnormally high blood pressure in all cases is not a defensible object *per se*. In certain cases, for example, when a cerebro-

[1] *The Lancet*, September 2, 1950.
[2] *The American Journal of Medicine*, March, 1950.

[1] *The Lancet*, September 2, 1950.

vascular accident is due to thrombosis, this reduction may be harmful, and it is not always possible to assess the individual patient. Moreover, as both Turner and Nickerson stress, we need much more information about the precise action of drugs that simulate the effects of sympathectomy. Their present use is largely empirical. The papers quoted can be added to reports of limited experience already published, such as that of C. C. Burt and A. J. P. Graham,[1] which most of our readers will already have seen, and that of P. Arnold and M. L. Rosenheim, whose experience was confined to the intravenous administration of pentamethonium iodide.[2] Turner has taken the clinical investigation of these drugs an appreciable distance further, and more reports of their use in experienced hands will be awaited with interest. They are obviously not yet for the tiro. If their clinical value in defined circumstances can be demonstrated satisfactorily, delay in their use while their precise action is determined may not be justified. It would be rash to suggest that the day of empiricism is dead.

MILK AND FOOD FOR SCHOOL CHILDREN.

Present proposals for the general supply of milk to school children will doubtless arouse problems quite apart from those associated with payment and distribution. Reports of English experience suggest that the children can provide just as many difficulties in the attainment of the original object of the plan as State Governments, and suggestions to flavour the milk in various exotic ways do not provide the whole answer. It may be helpful to take note of the English experience. According to Ffrangcon Roberts,[3] in a particular school in Cambridgeshire not more than 27% of the children can be made to drink milk. Admittedly, he adds, this figure is much lower than in neighbouring schools and lower than the average for the whole country, but the very mildness of this last comment suggests that the figure for the whole country cannot be very high. The attitude to milk in this particular school was linked with the fact that the school dinner, to judge by the amount left uneaten, appeared to be more than sufficient for the children, though it was well below the caloric standard recommended by the Ministry of Education. Roberts set out to investigate the position by a radiological study of the children's stomachs at intervals throughout the day. His findings are of considerable interest. At this stage it may save confusion to state that he uses the word "lunch" to describe the food that about half the children eat at the mid-morning break (10.35 to 10.45 a.m.) and dinner to describe the midday meal provided at the school. The other usual meals taken by the children were early morning tea (in many cases—sometimes in bed), a fairly substantial breakfast, tea (plain or high) at 4.30 p.m. and a substantial meal at 9 p.m. Milk time was at 10.35 a.m. Roberts found that at dinner-time all children who had taken milk with or without lunch two hours previously had a considerable quantity of food in the stomach; with one exception all said that they were not hungry. On the other hand, all children who took no milk had empty stomachs, and all said that they were hungry. As a general rule the children who habitually took no milk had empty stomachs at milk time, while those who habitually took milk had not completely digested their breakfasts. The desire for milk is therefore dictated, Roberts suggests, not by the emptiness of the stomach but by the habit of keeping the stomach filled. Later observation revealed that lunch without milk was digested more rapidly than milk without lunch. Whether milk had been taken or not, the children failed to eat a dinner of 788 Calories; the Ministry of Education's standard of 1000 Calories was apparently excessive for them. Advancing milk-time to 9.45 a.m.—that is, three hours before dinner—secured the attainment, desirable on physiological grounds, of an empty stomach by dinner-time; because of this it has been adopted as standard practice in the school, milk being distributed directly after assembly and prayers, though the change has not made milk more popular. The final examination for the day showed that whether milk had been taken or not, the stomach contained a quantity (sometimes large) of food four hours after dinner. Roberts's experience is, of course, confined to one school, but his findings will no doubt apply to some children elsewhere and may help in the understanding of the matter in Australia. He brings forward several reasons for the aversion to milk. During the war delivery was unreliable and it was not always possible to avoid mixing fresh milk with stale. Some children dislike cold milk, but mostly they dislike it whether warm or cold. Many parents do not insist on their drinking milk at home. Some girls have caught the mania for slimming from their elder sisters and regard milk as undesirable. Poverty is no reason, since milk has always been obtainable free in deserving cases. However, above all these reasons, in Roberts's opinion (supported, he states, by many experienced people whose judgement he respects) the chief reason for the refusal of milk is that children, at any rate those in the locality of the school investigated, are given in one form or another more food than they really need. That is a point well worth remembering. The willingness of most children to eat anything at any time of the day is often reinforced by parental encouragement. "Many mothers have a horror lest their children should feel the slightest pangs of hunger even for brief moments of the day." When milk is offered and a good dinner is provided, Roberts states (and most will agree), a packed lunch appears to be unnecessary. Thus, while few will suggest that the distribution of milk to children is not a desirable practice, it should not be allowed to cause undue heart burning if the children are not universally enthusiastic. Roberts emphasizes that he does not regard his findings as applicable to children all over England, but they may well apply widely in this country of abundant food.

THE SIGNIFICANCE OF THE CORTISONE DISCOVERIES.

Reports in medical journals and books of the effects of cortisone and adrenocorticotrophic hormone (ACTH) are accumulating so rapidly and bewilderingly as to form a flood, although it is only just over two years since Kendall's compound E became available to Hench and his co-workers and not much over a year since their first report appeared of its effect on rheumatoid arthritis. They would seem to have tapped a new artesian basin, and others have hastened to sink their own wells despite the expense of the operation. Many of the reports are significant and warrant review, but that is not our present purpose. It is rather to look back after this short but crowded interval at the significance of the original discovery as it has been pointed out by G. W. Pickering.[1] Professor Pickering has used an apt simile, different from our metaphor and more profound, in likening the discovery of Hench and his colleagues to Magellan's discovery of the straits that bear his name. For, Pickering goes on, although the existence of a vast ocean beyond the Americas had been known since the *conquistadores* crossed the continent, its exploration had been delayed by the absence of any known inlet from the seas of the Old World. The discovery of the effect of cortisone, and later of the similar effect of ACTH, not only on rheumatoid arthritis but on a variety of diseases of hitherto unexplained pathology, "seems to provide a means by which their pathology and pathogenesis will ultimately be solved". This, in Pickering's view, is the great significance of the discovery. As a background to the further discussion of this question, he considers broadly the causes of disease. He points out that the state in which the animal finds itself at any time is determined by two prime factors—its inherited constitution and the effects

[1] *British Medical Journal*, February 25, 1950.
[2] *The Lancet*, August 20, 1949.
[3] *The Lancet*, September 30, 1950.

[1] *The Lancet*, July 15, 1950.

of its environment, past and present—and lists the following common causes of disease: (i) inborn and inherited abnormalities; (ii) the effects of excess of a chemical agent in the environment; (iii) the effects of a deficiency of a chemical substance; (iv) infection or infestation by viruses, bacteria, fungi, or animal parasites; (v) physical trauma. A possible additional category would be "absence or deficiency of microbes that normally live in or on their host and contribute to his welfare"; as yet no known disease appears to correspond to this. Unfortunately the cause of a great number of diseases is not known; many may belong to one or several of the categories listed, but little or nothing is known of the causation of some conditions, including the rheumatic diseases, peptic ulcer and ulcerative colitis. Pickering refers to three hypotheses of disease that have been offered in this century to explain this last-mentioned group: focal sepsis, a psychosomatic origin and Selye's theory of diseases of adaptation. He gives short shrift to focal sepsis and the psychosomatic concept and suggests that "the small amount of critically established fact on which these two hypotheses were based can probably be accounted for on the fact that the condition of any patient suffering from a chronic disease deteriorates when there is superadded infection or disturbance of the mind". In this statement he might be regarded as giving back with one hand some measure of what he has taken away with the other, but further discussion of the point is not opportune here. The theory of diseases of adaptation, put forward by Hans Selye, of the University of Montreal, is one of great interest, and Pickering treats it with much more respect without being convinced. Selye's theory requires a separate consideration, which we will not attempt now. It is sufficient to state that he regards the hypophysis as of great importance in his adaptation syndrome, and thinks that it is above all the adrenal cortical secretions which produce changes on which resistance depends, and which are concerned in what he calls the diseases of adaptation. Pickering concedes that Selye's hypothesis is more solidly backed by experimental evidence than its predecessors, and also that it is made more plausible by Hench's quite independent discovery that one of the secretions of the adrenal gland would abolish many features of the pathological changes of rheumatoid arthritis. The great weakness, in Pickering's view, of the focal sepsis and psychosomatic hypotheses is the relative inefficacy of the therapeutic measures which each prompted. Cortisone (and, in general, ACTH, the work on which is less advanced) has been found to affect favourably rheumatic fever, rheumatoid arthritis, psoriasis, *lupus erythematosus*, *polyarteritis nodosa*, dermatomyositis, lymphatic leuchæmia, ulcerative colitis, bronchial asthma and urticaria. However, when he comes to consider how cortisone may act, Pickering finds difficulty in accepting the explanation that would derive from Selye's hypothesis: on it the diseases might be produced by excess of the mineralocorticoid secretion of the adrenal not balanced by a corresponding secretion of glucocorticoid; administration of cortisone would correct this disturbed equilibrium directly, and ACTH would correct it indirectly. Unfortunately, there appears to be no correspondence between these diseases and the effects of large doses of deoxycortone. Unless, therefore, Pickering states, the mineralocorticoid in question is one with properties very unlike deoxycortone, it cannot be accepted that overproduction of substances of this type produces these diseases. The possibility that these diseases are due to a total deficiency of suprarenal secretion can be ruled out, as they are "never or seldom" seen in association with Addison's disease. A more attractive possibility is that while the secretion of mineralocorticoids is normal, that of the glucocorticoids is diminished. Recent reports indicate that patients receiving cortisone in doses that produce remission in a variety of diseases sometimes develop features of Cushing's disease. It is not known whether continued administration of the smallest doses needed to produce remission will have this effect, but if so it seems unlikely, as Pickering states, that a simple deficiency of glucocorticoid secretion is a factor in pathogenesis. There is evidence that effective therapy requires a level of the hormone above normal. Pickering brings forward a number of other important points possibly associated with the action of cortisone and ACTH—they appear to affect profoundly tissue reactions of the body apart from the disease they relieve, and to affect certain kinds of tissue hypersensitivity, for example, the sensitivity to tuberculin; but he concludes that we still cannot account for the action of these substances by any of their effects as yet fully understood. What is clear, he states, is that "in these compounds we have new tools with which to attack some of the more obscure reactions of the body; and that the result of this attack may be the recognition of a new category in the causation of disease". That, vague generalization as it may appear to be, is a significant statement. It means that an important new door has been found and glimpses through the key-hole are encouraging; the lock has still to be mastered. Pickering emphasizes the fact that the discovery of cortisone was not a flash in the pan, but the result of a lifetime's use of the experimental method in the treatment of rheumatoid arthritis. There is much to be learnt yet in this field, and "new knowledge of the disease processes concerned may produce completely new remedies"; but the fundamental clues for the patient investigator seem to have been found.

RELIEF OF PAIN AND BOWEL ACTIVITY.

MORPHINE is probably the most popular and the most commonly used analgesic for the relief of post-operative pain, but its constipating effect has always been an important drawback, and this has been constantly in the mind of those seeking an alternative analgesic. An important contribution to the assessment of the effect upon bowel activity of various analgesics has recently been made by E. M. Vaughan Williams and D. H. P. Streeten.[1] They have devised an ingenious operation, the details of which need not concern us here, to isolate a segment of bowel, so that while it remains in the abdominal cavity of adults and retains its normal blood supply, it is accessible from the exterior for the introduction of drugs and for the recording of its activity. The amount of propulsive work done by this loop of bowel can be precisely estimated. Experiments were carried out on the effect on the bowel of morphine, pethidine and amidone, and it was found that whereas morphine and amidone in doses equivalent to those used clinically had a considerable inhibitory effect, pethidine, even if in much larger doses, did not interfere with intestinal propulsion. Vaughan Williams and Streeten state that it has been shown that there is considerable similarity between the reactions of human and canine gut; if the findings of the animal experiments are translated into terms of a human subject weighing 70 kilograms, it appears that whereas morphine in a dose of one-quarter of a grain would abolish propulsion of fluid for one hour or more, the same dose of amidone would be only just sufficient to cause a detectable inhibition, and pethidine in doses as high as 200 milligrammes would be well below the threshold for inhibition of propulsive activity. Clinical experience accords with these findings for these three fairly well-known drugs, and supports the view that the method should be of value in the assessment of newer analgesics.

INDEX TO "THE MEDICAL JOURNAL OF AUSTRALIA".

THE index to THE MEDICAL JOURNAL OF AUSTRALIA for the half-year ended June 30, 1950, has been published. Readers who are on the index mailing list will receive their copies as usual. Others who desire to receive a copy should apply to the Manager at The Printing House, Seamer Street, Glebe, New South Wales.

[1] *The Lancet*, August 5, 1950.

Abstracts from Medical Literature.

PATHOLOGY.

Parathyreoid Tumour with Visceral Metastases.

E. S. J. KING AND BARBARA WOOD (*The Journal of Pathology and Bacteriology*, January, 1950) describe a case of hyperparathyreoidism associated with a malignant tumour of the left lower parathyreoid gland in a woman aged forty-one years. After removal of this tumour the patient's bone condition was restored to normal, and biochemical studies showed a return to normal of the serum calcium, phosphorus and phosphatase levels. Recurrence of symptoms associated with the appearance of metastatic nodules in the lungs was soon followed by death. Post-mortem examination showed that there was no local recurrence, but that the lung deposits were composed of histologically typical parathyreoid tissue. So far as has been determined from a review of the literature, this is only the second recorded case of visceral metastasis of a proved parathyreoid growth.

The Pathogenesis of Non-Inflammatory Cerebral Aneurysms.

ROBERT CARMICHAEL (*The Journal of Pathology and Bacteriology*, January, 1950) states that two apparently contradictory views are held on the ætiology of non-inflammatory saccular aneurysms of the larger cerebral arteries; one ascribes them to degenerative lesions, the other to developmental medial defects. Both views are largely founded upon gross post-mortem findings. A fresh analysis of the relevant data, based partly on published work and partly on new material, shows a fairly even balance of evidence in favour of each of these views. The age distribution and the constant presence of thickened and degenerate intima in the sac suggest that degenerative disease is the main or even the sole causal agent. The virtually constant relationship of the aneurysms to the arterial bifurcations, where developmental defects occur, seems to warrant the view that developmental deficiency is the one essential basic lesion. The results of a histological study of 13 small aneurysms, cut in serial section, show that they all owe their origin to the combined effects of developmental deficiency and arterial degeneration. The aneurysms always arise at the site of substantial breaches in the muscular and elastic coats, and these coats are breached in different ways. The gap in the muscular layer is usually a focus of medial aplasia, which may be substantially enlarged by superimposed degenerative changes; but it is sometimes an area of hypoplasia in which the under-developed media has been destroyed by primary degeneration and fibrosis. So far as this layer is concerned, developmental deficiency is usually the dominant factor and degeneration plays only a subordinate part. But the gap in the elastic membrane is due to degenerative changes alone and chiefly to implication of the membrane in an ordinary atheromatous process, although other types of focal degeneration may help to widen the breach. The precise combination of lesions varies greatly from case to case, but both developmental and degenerative factors are concerned in the genesis of all these aneurysms, and no valid distinction can be made between the so-called congenital (that is, developmental) and arteriosclerotic types. The basic lesions in the intima and media seem to affect the same part of the arterial wall only by chance, and this is probably the principal reason for the relatively low incidence of the aneurysms. But the sac apparently requires an outlet through the media comparable in size with the larger defects found at normal bifurcations, together with a gap of similar dimensions in the internal elastic lamina. These requirements and the relatively resistant character of some atheromatous plaques are additional factors tending to limit the incidence of cerebral aneurysms.

Primary Vascular Changes in the Lungs.

H. SPENCER (*The Journal of Pathology and Bacteriology*, January, 1950) describes how four cases of primary vascular disease of the lungs have shown the existence of at least two separate pathological conditions associated with the clinical condition of primary pulmonary hypertension. The first two cases showed changes leading to complete occlusion of the smaller muscular branches of the pulmonary artery by obliterative endarteritis. This change was probably initiated by congenital weakness of the walls of the smaller muscular pulmonary arteries, with compensatory intimal proliferation and arterio-venous anastomosis. The second group of two cases showed an entirely different picture, namely, that of chronic, mainly perivascular, interstitial pneumonia of unknown ætiology. This group was characterized by perivascular lymphocytic cuffing of the smallest pulmonary arteries and arterioles. Later, severe perivascular fibrosis and *endarteritis obliterans* caused obstruction to the pulmonary blood flow. In addition to these four cases, two further cases are discussed illustrating two other primary pulmonary vascular disorders, unassociated with pulmonary hypertension, and the possibility is suggested of these two conditions causing pulmonary hypertension. The necessity is stressed for widespread thrombosis of the pulmonary arterial tree if pulmonary hypertension is to result. The possible association of medial hyaline degeneration of pulmonary arteries and hypersensitivity to a pneumococcal lung infection is suggested by the occurrence of similar changes in cerebral vessels in pneumococcal meningitis.

Plasma-Cell Tumours.

A. J. RAWSON, P. W. EYLER AND R. C. HORN (*The American Journal of Pathology*, May, 1950) have studied the histological characteristics of nine cases of plasma-cell tumour involving the upper parts of the respiratory and food passages (six malignant neoplasms and three benign or inflammatory lesions) and have compared them with the histological picture of nine cases of multiple myeloma and with a variety of inflammatory lesions in which plasma cells were an especially conspicuous feature. They state that the replacement of tissue by broad sheets of plasma cells oriented on a delicate capillary stroma is indicative of a malignant plasma-cell tumour. The absence of this pattern makes it unlikely that a plasma-cell tumour is malignant. The usual cytological criteria of malignancy are reliable in the study of plasma-cell tumours when they are present in considerable degree. The absence of these criteria, however, does not exclude malignancy. Benign plasma-cell lesions of the upper part of the respiratory tract may be adequately treated by surgical excision. The malignant plasma-cell neoplasms respond well to radiotherapy; in the cases reported, recurrence was the rule when treatment was by surgical removal.

Fat Embolism in Trauma.

J. P. WYATT AND P. KHOO (*American Journal of Clinical Pathology*, July, 1950) have studied a consecutive series of 30 subjects who died after trauma, to determine the frequency and amount of intravascular fat embolism. In every instance evidence of intravascular pulmonary fat was present. A similar study in patients who died suddenly from non-traumatic causes or from lingering illnesses did not show notable amounts of intravascular fat.

Pleural Mesothelioma.

WILLIAM N. CAMPBELL (*The American Journal of Pathology*, May, 1950) states that the diagnosis of pleural mesothelioma was once held in ill repute by pathologists. The failure to accept pleural mesothelioma as a distinct entity was due mainly to a lack of understanding of the neoplastic multipotentialities of the mesothelial cells of the pleura. In addition, many so-called primary pleural tumours were shown by careful gross and cytological scrutiny to have been metastatic from somewhere else in the body, the usual primary site being the bronchus. As a consequence it is probable that many true pleural mesotheliomata have been unrecognized, pathologists being overcautious in making such a diagnosis. The author suggests, on the basis of a study of four new cases of pleural mesothelioma, verified by necropsy, and of 10 recently reported cases, that a definite diagnosis of pleural mesothelioma can be made in the majority of cases on cytological grounds alone, since most of these 14 tumours, and many of the tumours in the older literature, exhibited a cyto-architecture that was epithelial in appearance in some areas and mesenchymal in others. The characteristic method of spread of pleural mesothelioma was found to be by contiguity and serosal seeding; this should help differentiate the process from bronchogenic carcinoma. Since pleural mesotheliomata grow rapidly, a plea is made for early diagnosis in the hope that early radical surgical extirpation may result in cure.

A Distinctive Vacuolar Nephropathy Associated with Intestinal Disease.

J. P. KULKA, C. M. PEARSON AND S. L. ROBBINS (*The American Journal of Pathology, May, 1950*) have studied 15 cases of a distinctive nephropathy associated with chronic intestinal disease. This condition was found in eight of 72 cases of non-specific ulcerative colitis, as well as in seven of 80 cases of intestinal disease of various other types. The lesions were characterized by the presence in the

proximal convoluted tubular epithelium of well-defined, clear vacuoles, which were frequently so large that they "ballooned out" the cells which contained them. These vacuoles failed to stain by any of the usual methods, including those for fat and glycogen. The nuclei were generally displaced toward the base of the cells, but remained relatively well preserved. The distribution of the vacuoles was patchy, affecting some tubules more than others and frequently involving certain cells and sparing adjacent ones in the same tubule. The clinical findings in these cases did not conform to a consistent pattern. Even intestinal symptoms were not always significant. Nevertheless, the cases were similar in that all of the patients had been ill for more than four weeks, and, at least terminally, they probably all developed some degree of malnutrition, anæmia, hypoproteinæmia, electrolyte imbalance and sepsis. Signs and symptoms of kidney dysfunction were varied and inconstant, and failed to correlate with the severity of the vacuolation. Certain presenting nephropathic factors may have pathogenic significance. The authors consider that although the ætiology and pathogenesis of these renal lesions remain obscure, the coexistence of intestinal disturbances with these tubular changes suggests a possible causal relationship.

Arteritis of the Appendix.

JOHN W. HALL, SHAO-CHIEN SUN AND WILLIAM MACKLER (*Archives of Pathology*, August, 1950) state that arteritis of the appendiceal arteries and arterioles is an occasional finding in an otherwise normal appendix of a patient who has the signs and symptoms of acute appendicitis. The arterial lesion is possibly allergic in origin, representing either a focal allergic reaction to bacteria or bacterial products or a local manifestation of a generalized vascular lesion resembling *polyarteritis nodosa*.

MORPHOLOGY.

Unusual Anomaly of Flexor *Digitorum Longus* Muscle.

T. E. BARLOW (*Journal of Anatomy*, July, 1949) describes an interesting anomaly of the *flexor digitorum longus* muscle in the foot and adduces a reason for its presence. The interest in this case lies in the support it gives to the phylogenetic history of the flexor muscles of the toes as interpreted by Wood Jones.

Œstrogen and Anterior Hypophysis.

J. M. WOLFE (*American Journal of Anatomy*, September, 1949) studied the cellular changes in the anterior hypophyseal lobe of the rat induced by the administration of œstrogen, using newer cytochemical methods which made it possible to identify with considerable exactness certain chemical substances in the cell, such as nucleoproteins, certain enzymes and mucoproteins. The purpose was to study the role of nucleoproteins in secretory phenomena in anterior lobe cells and to compare and correlate findings obtained with more or less traditional cytological procedures which demonstrate secretory granules, mitochondria and the Golgi bodies. Immature female rats received 33 microgrammes of α-œstradiol benzoate for periods of from ten to thirty days. The anterior lobes of untreated sisters and of normal and castrated male rats were used as controls. Œstrogen administration induced pronounced secretory changes in the acidophile and chromophobe cells: there was evidence of the stimulation of the elaboration and release of secretion; but observations on the basophile cells were limited and the findings were more variable and less distinct. Changes considered to be retrogressive in nature were observed in the chromophobe cells, and these changes were thought to be characteristic of formerly actively secreting chromophobe cells undergoing transformation into inactive chromophobe cells.

Nerve Implants in Voluntary Muscle.

J. T. AITKEN (*Journal of Anatomy*, January, 1950) describes a method of implanting nerves in muscle with minimum injury to the muscle. He states that nerves which regenerated into a denervated muscle formed functioning motor end-plates, while nerves which regenerated into normal muscle rarely formed motor end-plates, and these were probably on muscle fibres that had been damaged at operation. In the implants in denervated muscles more than one ending was frequently seen on a muscle fibre. Implants into tenotomized muscles formed long thin fibres, which travelled between the muscle fibres and occasionally coiled round them. No regenerated endings were seen. The non-receptiveness of normal muscle fibres is discussed.

Effect of Electric Shock on Brain.

R. G. SIEKERT *et alii* (*Archives of Neurology and Psychiatry*, January, 1950) have restudied the problem of whether electric shock therapy in the treatment of certain mental disorders produces histological changes in the central nervous system. They state that numerous investigators had previously studied this problem, but it had not been conclusively shown that changes did occur. In the first investigation five monkeys were given courses of electric shock treatments comparable to those used in human convulsion therapy. An additional monkey served as a control. After the last treatments of the experimental animals, all were killed and the brains fixed by an improved technique of intravascular perfusion and fixation to prevent the development of post-mortem artefacts in the brain and spinal cord. Histological study of sections of the central nervous system stained by precisely controlled neurological methods revealed no difference between the experimental and the control animals. There were no neuronal changes, glial proliferation, areas of demyelination or evidence of old or recent hæmorrhages in either series. No structural alteration was demonstrated after experimental electric shock.

Sympathetic Contributions to Roots of Brachial Plexus.

S. SUNDERLAND AND G. M. BEDBROOK (*Brain, September, 1949*) report estimates of the relative percentage contribution of post-ganglionic sympathetic fibres to each root of the brachial plexus in man. Four subjects were examined, and considerable variation was found. The highest percentage was found in each case in the eighth cervical root (25% to 45%) and the lowest in the fifth cervical (1% to 9%). Other results were as follows: sixth cervical (8% to 27%), seventh cervical (15% to 25%), first thoracic (15% to 28%); 40% to 70% of post-ganglionic fibres were contained in the lower trunk.

Efferent Connexions of Frontal Lobe.

M. MEYER (*Brain*, September, 1949) states that he has examined brains from patients with a short survival time after prefrontal leucotomy to trace cortico-fugal fibres to their termination in subcortical nuclei. A detailed investigation of cortico-fugal fibres undertaken by modern methods has not previously been reported for the human brain. After the production of lesions restricted to the prefrontal cortex, a few fibres terminate in the dorso-medial nucleus of the thalamus, but the assertion based on experimental findings that the prefrontal cortex projects to other thalamic nuclei or to extrapyramidal nuclei is not confirmed for the human brain. After the production of lesions extending to the white matter which underlies Brodman's areas 6, 8 and 9, terminal degeneration is present in the mamillary body, dorso-medial and ventro-lateral group of nuclei of the thalamus, *globus pallidus, zona incerta*, Forel's field, red nucleus and subthalamic nucleus. The finding of a direct cortico-pallidal pathway is of particular interest, as this has been a matter of considerable controversy. Although it has been well established by animal experimental work that the anterior nucleus of the thalamus projects to the cingulate gyrus, in this investigation no reciprocal cortico-fugal connexions could be demonstrated from the rostral part of the cingular gyrus to the anterior nucleus of the thalamus.

Changes in Hypophysis during Growth Period.

A. T. RASMUSSEN (*American Journal of Anatomy*, January, 1950) states that he has examined histologically the normal hypophysis of 72 males and 66 females from birth to nineteen years of age. Many features of the adult hypophysis are already established at birth. At birth the residual lumen is always present and the *pars intermedia is a more uniform stratum than later* in life. Calcified concretions were found in fully half of the specimens, and the incidence of concretions within the anterior lobe was greater during the first six months of life. No significance is attached to these benign structures. The average number of basophile cells at birth is about 9% of all the cells. With age there is a slight increase in males and a slight decrease in females, but individual variation is considerable. The acidophile cells at birth represent about 25% in males and 29% in females, but there is a noticeable increase with age in both sexes. At the age of nineteen years the numbers are 40% and 49% respectively, so there are 9% more acidophile cells in females than in males. Chromophobe cells represent 61% at birth in females and 65% in males, decreasing with age to 43% and 48% respectively at the age of nineteen years. The results found in this investigation do not indicate the existence of distinct histological cycles that can be correlated with physiological cycles.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on July 27, 1950, at the Robert H. Todd Assembly Hall, British Medical Association House, 135 Macquarie Street, Sydney, DR. G. C. HALLIDAY, the President, in the chair.

The Indications for Splenectomy.

DR. C. R. B. BLACKBURN read a paper entitled "The Indications for Splenectomy" (see page 641).

DR. W. L. CALOV read a paper entitled "The Indications for Splenectomy" (see page 644).

DR. A. H. TEBBUTT said that he did not think that the indications for splenectomy were always so nicely considered as they had been by the speakers. Some of the most striking and largest spleens had not been removed for the conditions which were found to be present. The usual indication for splenectomy had been Banti's disease. Dr. Tebbutt remembered a very large spleen which was removed for Banti's disease; the condition proved to be sarcoidosis. He had also seen a spleen removed in Hodgkin's disease, but not under that diagnosis. Still other spleens had in his experience been removed for conditions which proved to be myeloid metaplasia. Dr. Tebbutt thought that physicians should consider the diagnosis of myeloid metaplasia at times; he had not known a case in which that diagnosis had been made before operation or death. The conditions presented by the patients were anæmia, a very large spleen and a blood picture suggesting leuchæmia—not manifest leuchæmia, but certainly a considerable "shift to the left". Dr. Tebbutt said that this was the condition in which, as Dr. Blackburn had mentioned, splenectomy was contraindicated, because the blood cells were being produced by the spleen instead of or as well as by the bone marrow. Dr. Tebbutt did not know whether that was universally true, and wondered whether Dr. Blackburn would have something to say on the subject. Was the condition always myelofibrosis? Did myeloid metaplasia always follow? Dr. Tebbutt said that in the two cases he had mentioned, one of the patients died, and the other survived and was still alive with a myeloid blood picture a year or so after operation, although the operation was said to be contraindicated. The use of X rays was also contraindicated in myeloid metaplasia. Dr. Tebbutt said that he had one more point to raise, in which perhaps Dr. Blackburn could help him. Dr. Blackburn had shown a slide illustrating diagrammatically aplastic anæmia; did he mean pancytopenia from hypersplenism, which presented a blood picture of aplastic anæmia? Dr. Tebbutt assumed that it was not aplastic anæmia in the strict sense, and that the marrow was cellular. Referring to the differential diagnosis of pancytopenia from aplastic anæmia, Dr. Tebbutt said that Bomford and Rhoads in their paper on refractory anæmia had described one variety of aplastic anæmia which was not aplastic in the ordinary sense—the marrow was active. Dr. Tebbutt asked Dr. Blackburn whether he would consider such a condition as possibly pancytopenia due to hypersplenism. Dr. Tebbutt did not know that Bomford and Rhoads had considered hypersplenism, their interest being only in the causes of refractory anæmia, and whether such anæmia was due to chemical or physical noxious substances. In many cases they were unable to incriminate anything. Dr. Tebbutt wondered whether refractory anæmia in some of Bomford and Rhoads's cases could be due to pancytopenia, not considered at that time. Could it be said in any case that pancytopenia was due to hypersplenism, or that it was either idiopathic or due to some chemical or physical depressant?

DR. E. F. THOMSON said that the subject of the spleen and its functions and dysfunctions, as had been shown by the two papers read, was one of extraordinary interest. He proposed to mention three cases, which showed that the question of whether to remove the spleen or not was not easily answered. One case in particular required comment from Dr. Blackburn. An elderly man had been admitted to the Royal Prince Alfred Hospital presenting the classical picture of thrombocytopenic purpura. He was rushed to operation and subsequently died. The removed spleen was found to be the site of an enormous tumour, the nature of which had not yet been determined. The second case was that of a woman who had hæmolytic anæmia, with a positive response to the Coombs test. Her spleen was removed, the hæmolytic process persisted, and she died. The third case was that of a young woman who was admitted to hospital with the typical picture of idiopathic thrombocytopenic purpura. Her spleen was removed and she made a miraculous recovery. Dr. Thomson asked Dr. Blackburn to comment on the tumour of the spleen in the case in which everyone thought the diagnosis was thrombocytopenic purpura.

DR. A. C. THOMAS said that his interest in splenectomy lay in the fact that he did not wish to regard himself as a carpenter, in performing the operation only at the request of a physician, but wished to acquaint himself with the rationale behind the procedure; that was why he was present. A number of ruptured spleens were encountered, and the patients did remarkably well. Recently he had had to perform splenectomy, at the request of a senior physician, on a patient who had aplastic anæmia of the type that had been discussed earlier in the meeting. He (Dr. Thomas) had read in the *British Medical Journal* an account of a lecture, delivered at Belfast by a surgeon of Saint Bartholomew's Hospital, London, elaborating Dameshek's theory of hypersplenism. The patient had been examined only a few months prior to the meeting; he was a man presenting a typical picture of aplastic anæmia; he had had numerous blood transfusions, but almost as fast as they were given the blood picture returned to that of a very grave anæmia, the red cell count falling to about 1,000,000 per cubic millimetre. A consulting physician had asked him (Dr. Thomas) to perform splenectomy, which he did. The spleen was rather large and had many peripheral adhesions, but the patient did remarkably well. Blood transfusions were given at operation and immediately afterwards, and the blood findings quickly attained a respectable figure. The patient had required no further blood transfusions. A few years previously Dr. Thomas had performed splenectomy at the request of a physician on a patient presenting a similar picture of aplastic anæmia and requiring blood transfusions every week. After operation the patient continued to require blood transfusions, but at intervals of six weeks instead of one week. Dr. Thomas said that he could not add to what had been said about the theory of the conditions under discussion; but he believed that when a surgeon performed splenectomy, he should try to know what he was doing. He himself had performed splenectomy on a number of patients suffering from thrombocytopenic purpura, and they had done remarkably well. Another patient suffered from the acquired type of hæmolytic acholuric jaundice; she had been under treatment for many years for a variety of vague abdominal conditions. The diagnosis was interesting in that the patient had come into the care of a general practitioner, who, looking for a cause of her disorder, had a blood count made; some spherocytes were found. Further investigations by means of fragility and other tests showed the patient to be suffering from hæmolytic acholuric jaundice. Her spleen was removed and she did remarkably well, but her blood picture remained unchanged; spherocytes were still present. Dr. Thomas congratulated the speakers on the high standard of their papers and said that he had profited from listening to them and to the ensuing discussion.

DR. R. H. BLACK said that malaria was a subject that might be further explored at the meeting. One encountered cases of subcapsular hæmorrhage which raised the question whether the spleen was spontaneously ruptured. Such cases presented a problem in the decision whether the spleen required surgical removal. Andrew had reported a series associated with *Plasmodium vivax* malaria. The splenic condition settled down with conservative treatment. One was faced with an urgent decision; the occurrence of such cases should be borne in mind.

Dr. Blackburn, in reply to Dr. Tebbutt's first question (whether myeloid metaplasia was always associated with generalized myeloid fibrosis), said that in his paper he was not referring specifically to myeloid metaplasia. What he had meant to say was that if a patient had only his spleen to depend on for blood formation—for example, in myelofibrosis—then splenectomy was not indicated. In such a case all the patient's blood formation might be taking place in the spleen. But it was impossible to say, from the examination of one piece of bone marrow, that all the blood formation was taking place outside the marrow. Referring to the diagrammatic slide of aplastic anæmia which he had shown, Dr. Blackburn said that it illustrated a marrow that was not producing cells for any reason; he had not intended to make any point about "aplastic anæmia", because he found difficulty in defining it. In reply to Dr. Tebbutt's third question, Dr. Blackburn referred to a series of cases of "aplastic anæmia" reported by W. P. Thompson *et alii* at the Presbyterian Hospital, New York; those authors had stated that if they were to restrict the diagnosis to patients who presented all the stigmata of

aplastic anæmia, they had seen no cases in the past fifteen years. The point about the diagnosis of aplastic anæmia was that, if one insisted that all the criteria should be there, it would be an exceedingly rare disease. In the Clinical Research Unit at Royal Prince Alfred Hospital there had been one patient who nearly fulfilled all the criteria, but the total number of reticulocytes was within normal limits. The condition to which he had referred in the diagram was really one in which the marrow was extremely hypoplastic. With regard to the first case quoted by Dr. Thomson, that of an elderly man suffering from idiopathic thrombocytopenic purpura, Dr. Blackburn said that such a description seemed almost a contradiction in terms; an elderly man was the rarest type to suffer from that disease and a "cause" was found. The obvious way out of the difficulty was to say that the case was one of hypersplenism particularly affecting the production of platelets. That was the way out which Dr. Blackburn proposed to take. Referring to Dr. Thomson's second patient, a woman who gave a positive response to the Coombs test, Dr. Blackburn said that the case illustrated the situation in which all the antibody coating the cells was not being produced in the spleen. The same comment on his cases of aplastic anæmia further illustrated how difficult the condition was to diagnose. A patient who required blood transfusions at intervals of one week was destroying red cells at a very rapid rate. That was commonly seen in so-called aplastic anæmia, but might be missed if no study was undertaken of the life span of the red cells (the normal span was about 120 days).

Dr. Calov, in reply, said that he had been interested in the remarks on aplastic anæmia made by Dr. Tebbutt and Dr. Thomas. He said that from his own reading he understood that in some cases of so-called aplastic anæmia, when all the other signs of so-called aplastic anæmia were present, one might take a sample of the marrow and find it hypoplastic, another sample and find it hyperactive, and yet another sample and find it normal. Perhaps his interpretation of what he had read was wrong. If he was right, then he thought that the term "aplastic anæmia" should be abandoned. In the cases mentioned by Dr. Thomson and also in those mentioned by Dr. Thomas, blood transfusions had been given. Dr. Calov said that he did not pretend to know what happened in cases in which repeated blood transfusion was given; but he knew that death sometimes occurred after a blood transfusion had been given. He well remembered one woman with a huge spleen who had undergone splenectomy on his recommendation; the operation was satisfactory and there seemed to be no necessity to do anything more. Then somebody gave her a transfusion of a litre of blood, and not long afterwards she died. Dr. Calov thought that medical practitioners should be much more careful than they were about giving blood transfusions. Dr. Calov went on to say that the remarks about subcapsular hæmorrhage had been interesting. He had examined natives who appeared to have a rupture of the spleen. On one occasion someone had kicked a football at a native and hit him in the belly. The native was in great pain and suffering from shock. Dr. Calov had made a diagnosis of rupture of the spleen and had decided to operate on him, but on the advice of Dr. G. A. M. Heydon delayed the operation. The patient recovered. Dr. Calov thought that that was a case of subcapsular hæmorrhage. The enlarged spleen in malaria was very big and spongy, and if such a spleen ruptured the effect was the same as if the patient had been taken by a shark.

A MEETING of the South Australian Branch of the British Medical Association was held on September 28, 1950, at the Verco Theatre, University of Adelaide, DR. C. O. F. RIEGER, the President, in the chair.

Recent Developments in the Study of Virus and Rickettsial Infections.

MISS NANCY ATKINSON read a paper in which she discussed some recent developments in the study of virus and rickettsial infections. She said that in recent years there had been much expansion in virus and rickettsial work. Numerous microbiological laboratories were introducing extensive work in newly built virus units designed and equipped to minimize the risk to workers and outsiders from the highly infectious material within. Notable among such virus laboratories was the new infectious disease building at the National Institute of Health, Bethesda, Maryland, United States of America, which she had visited during her recent study leave abroad.

Miss Atkinson went on to say that not only had virus laboratories been greatly improved, but also improvement had been achieved in techniques for handling viruses and rickettsiæ. For example, it was now known that most viruses might be preserved for months or years in deep freeze cabinets, refrigerated either electrically or with solid carbon dioxide. Purification of virus or rickettsial material was greatly assisted by chemical treatments, such as ether extraction, or by differential centrifugation by means of the now commercially available 18,000 and 40,000 revolutions per minute centrifuges, which had been so scarce as to be almost unknown a few years ago. Electron microscopy was revealing the structure of viruses and rickettsiæ, and cultivation in the chick embryo was rapidly being applied to most rickettsiæ and many viruses; they were thus rendered more accessible to experimentation.

Diagnostic tests, especially serological tests, had been developed for numerous virus and rickettsial infections. The success of such tests had depended upon the discovery of suitable antigens. Complement-fixing antigens for neurotropic viruses, rickettsiæ and other viruses had been developed, often from chick embryo material, and their specificity and anticomplementary activity had been rendered satisfactory by treatment with acetone, ether or some other solvent. The discovery of the hæmagglutinating power of various viruses had allowed the introduction of another serological test, known as hæmagglutination inhibition. In the United States of America such serological work was made easier by the commercial availability of some of the commoner virus antigens.

Miss Atkinson further said that a stimulus to virus work had also come in the commercial world through the large programmes instituted by many of the large chemical and biological manufacturing companies to try to discover new antibiotics and chemotherapeutic agents. The action of any new substance on viruses and rickettsiæ was of paramount importance, and tests were therefore included in any comprehensive scheme, such as that of Imperial Chemical Industries in England. Another development was the introduction of world centres for the study of certain epidemic viruses. The influenza centre was at the National Institute for Medical Research at Mill Hill, London, where Dr. Andrewes, who was in charge, hoped to acquire a complete range of influenza virus types to be used for prophylactic vaccination in epidemic times. Extensive investigation of the occurrence of "Q" fever in the United States of America had shown that thousands of cases had been occurring unrecognized in California, and the rickettsiæ had been found in milk, cream and butter, besides other materials from cattle. That work was of interest in South Australia, where "Q" fever had not long been recognized.

Miss Atkinson said that the neurotropic viruses interested her very much. In the United States of America she had seen some of the work on the typing of poliomyelitis virus in monkeys, carried on under the National Foundation for Infantile Paralysis. She had also been introduced to the neurotropic viruses maintained at the Rockefeller Institute in New York City, for which no natural disease had yet been found; so far only the experimental disease in mice is known. Those viruses were found in insects, native animals and other materials collected during the yellow fever campaign in the jungles of Africa. She had also seen a great deal of work going on with the new Coxsackie viruses, of which a number of different serological types had already been found. In Adelaide they were just commencing work on material from the recent poliomyelitis epidemic. At present they were searching for Coxsackie virus, and in suitable cases for mumps virus. They had an isolation unit incorporating the essential features of the large laboratories overseas, and they hoped soon to have some interesting results.

Correspondence.

ACROPATHY, ACRODYNIA INFANTILIS (FEER), PINK DISEASE (SWIFT) AND THEIR DIFFERENTIAL DIAGNOSIS AS TO USTILAGINISM (MAYERHOFER).

SIR: The valuable contribution from Professor Mayerhofer which appeared in your journal on October 7 will be of great interest to all who are striving to solve the ætiological problems of pink disease. Reference to your report of a local discussion published on November 18, 1939, will prove

that the work of Professor Mayerhofer and his collaborators was traversed then by me and that I was careful to state *inter alia* that "Mayerhofer and Dragasic had themselves made it quite clear that they knew Selter-Swift-Feer disease and that it occurred, too, in Yugoslavia. They differentiated clearly between the two conditions [pink disease and ustilaginism] and did not suggest that they overlapped nor should be confused with each other".

Yours, etc.,

H. BOYD GRAHAM

"Chanonry",
14 Collins Street,
Melbourne.
October 11, 1950.

"NEW AUSTRALIAN" DOCTORS IN MIGRANT CAMPS.

SIR: Quite a number of "New Australians" are medical men with European degrees and specialist diplomas. These men and women have come to Australia, knowing that they will have to do the professional part of our medical course. Most of them will have to work and save in order to pay their way. They are people with a culture, whose impact cannot but have a beneficial effect upon us and our profession, assuming that we welcome them. The production of "La Tosca" at Bathurst, Young and other country centres has already brought to our notice one who is a brilliant musician (a doctor of music) as well as an oculist.

Most of us know how easy it is for professional people to become intolerably lonely, and this is particularly so under camp conditions, where contacts with their own professional kind are not easy to find.

Some of us feel that we of the medical profesison should welcome them and encourage them to win through, ultimately becoming, not only Australians, but a stimulus to ourselves. It would be a good thing if, on their arrival at their camps in Australia, we as a corporate body could welcome them in writing. Also, our text-books and journals would assist them to keep medically alert, to acquire a fuller knowledge of English, to learn the Australian approach, and to encourage them to win through.

Mr. J. H. Blackert, of Kendal Street, Cowra, with the help of Dr. Alec McLaren and Dr. Thomas Atkins, both of Cowra, is quite willing to undertake the distribution of text-books and journals to the medical men in the camps at Cowra, Bathurst and Parkes centres.

Yours, etc.,

ALFRED J. STOCKS, M.B., Ch.M.

"Woorinyan",
Lovell Street,
Young.
October 5, 1950.

The Royal Australasian College of Physicians.

ANNUAL MEETING, 1951.

THE next annual meeting of The Royal Australasian College of Physicians will be held at Sydney from Wednesday, April 11, to Saturday, April 14, 1951.

EXAMINATION FOR MEMBERSHIP.

AN examination for membership of The Royal Australasian College of Physicians will take place in March-April, 1951. The written examination will be held in capital cities of the Commonwealth where candidates are offering. The clinical examination will be held in Sydney. Only those candidates whose answers in the written examination have attained a satisfactory standard will be allowed to proceed to the clinical examination.

The dates of the examination will be as follows: written examination (capital cities), Saturday, March 3, 1951; clinical examination (Sydney), approximately April 6 to 11, 1951.

Applications to appear before the Board of Censors should be made on the prescribed form, and must be in the hands of the Honorary Secretary of the College not later than Saturday, February 3, 1951. Candidates should signify in which city they desire to take the written examination. Application forms are obtainable from the Honorary Secretary, 145 Macquarie Street, Sydney.

LECTURE BY SIR CHARLES SYMONDS.

AN invitation is extended to all members of the medical profession to attend a lecture to be given under the ægis of The Royal Australasian College of Physicians by Sir Charles Symonds, K.B.E., C.B., M.D. (Oxon.), F.R.C.P. (Lond.), M.R.C.S., physician in charge of the Department of Nervous Diseases at Guy's Hospital, London. The lecture will be held on Monday, November 6, 1950, at 8.30 p.m., in the Stawell Hall of The Royal Australasian College of Physicians. The subject will be "Migrainous Variants".

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Week-End Course at Bega.

THE Post-Graduate Committee in Medicine in the University of Sydney, in conjunction with the Far South Coast and Tablelands Association, will hold a week-end course at the Bega Valley County Council Rooms, Auckland Street, Bega, on Saturday and Sunday, November 18 and 19, 1950. The programme is as follows:

Saturday, November 18: 2 p.m., welcome to visitors by chairman; 2.15 p.m., "Diabetic Coma", Dr. F. A. E. Lawes; 4 p.m., "Recent Advances in Ear, Nose and Throat Work", Dr. Bernard B. Blomfield.

Sunday, November 19: 9.30 a.m., "Recent Advances in Therapeutics", Dr. F. A. E. Lawes; 11 a.m., "Sinusitis", Dr. Bernard B. Blomfield.

The fee for attendance will be £2 2s. Those wishing to attend are requested to communicate with Dr. F. J. Ireland, Honorary Secretary, Far South Coast and Tablelands Association, Gipps Street, Bega, 7C., as soon as possible.

Congresses.

THIRD INTERNATIONAL CONGRESS OF LEGAL AND SOCIAL MEDICINE.

THE Third International Congress of Social and Legal Medicine will be held in Paris from September 23 to 28, 1951. It is proposed to hold a world-wide symposium in which important problems of social and legal medicine will be discussed. The President of the congress is Professor R. Piedelievre, a Member of the Academy of Medicine. All members of the medical profession are invited to attend. Further information may be obtained from the secretary-general, Professor Agrege Derobert, 1 rue Clovis, Paris (5e).

Obituary.

EDMUND HAROLD MOLESWORTH.

WE regret to announce the death of Dr. Edmund Harold Molesworth, which occurred on October 16, 1950, at Bellevue Hill, Sydney.

JAMES LISTER WHITWORTH.

WE regret to announce the death of Dr. James Lister Whitworth, which occurred on August 4, 1950, at Melbourne.

CHARLES THOMAS BOODLE MAISEY.

We regret to announce the death of Dr. Charles Thomas Boodle Maisey, which occurred on October 14, 1950, at Kingsford, Sydney.

BERTRAM LINDSAY MIDDLETON.

We regret to announce the death of Dr. Bertram Lindsay Middleton, which occurred on October 16, 1950, at Murrurundi, New South Wales.

University Intelligence.

THE UNIVERSITY OF MELBOURNE.

The following information is taken from the *University of Melbourne Gazette* of October 9, 1950. The Gazette is published by the Registrar for the Council of the University.

Dr. D. P. Madigan has been awarded the David Grant Scholarship for 1950, consisting of a medal and the sum of £70.

Miss Jean Millis, formerly senior lecturer in biochemistry, left Melbourne on September 22 to take up her appointment as Lecturer in Applied Nutrition in the Department of Social Medicine and Hygiene in the University of Malaya.

At a recent meeting of the Faculty of Medicine, Mr. K. Burnside was reappointed Stewart Scholar in Surgery, Mr. H. C. Trumble, Guy Miller Tutor in Surgery, Dr. W. M. Lemmon was appointed Thalia Roche Demonstrator in Obstetrics, and Dr. Kate Campbell, Clinical Lecturer in Infant Welfare.

As a result of recent negotiations between the Australian Vice-Chancellors' Committee and the Orient Lloyd Travel Service Proprietary, Limited, and through the generosity of the Australian National Airways, concessions in relation to air travel may be made to university scholars going from Australia to Great Britain and continuing their journey, within fifteen months, via New York and San Francisco, back to Sydney. Bookings should be made through the Orient Lloyd Travel Service in Melbourne, and applications for concession will be referred through the local Vice-Chancellor to the Vice-Chancellors' Committee.

The Vera Scantlebury Brown Memorial Appeal continues to make good progress, and a recent Press note indicated that £18,000 had been raised towards the objective of £25,000.

THE UNIVERSITY OF SYDNEY.

The University of Sydney has published an official journal: *The Gazette, University of Sydney.* In the first number the Vice-Chancellor, Professor S. H. Roberts, states the objects of the venture as follows:

> Firstly, we want to keep the great graduate body informed about University happenings. Secondly, we desire to let our staff members and students become as fully informed as possible and to counteract any tendency towards undue segregation in Faculties or Departments. Thirdly, we wish to let sister Universities know about our activities in this current period of academic stress and strain. Fourthly, since we are appealing to the public for support on quite a new scale, it is only fair that the community should learn in detail about University activities and trends, and hear about our positive achievements as well as our difficulties and worries.

The intention is to "publicize challenging and provocative statements by local and visiting experts in all phases of tertiary education". At the same time factual statements appearing in *The Gazette* about university happenings will be official. Copies of *The Gazette* will be sent to anyone sending name and address to the Registrar.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 7, 1950.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.[2]	Australian Capital Territory.[2]	Australia.[2]
Ankylostomiasis	*	..	..	..	..	..	..	..	..
Anthrax	*	..	..	..	..	..	..	..	..
Beriberi	*	*	*	*	*	*	..	*	..
Bilharziasis	*	..	..	..	..	..	..	..	..
Cerebro-spinal Meningitis	1(1)	1(1)	3(1)	..	..	..	..	..	5
Cholera	..	..	..	..	..	..	..	..	..
Coastal Fever(*a*)	*	*	..	*	*	*	*	*	..
Dengue	*	*	*	*	*	*	..	*	..
Diarrhœa (Infantile)	..	..	1(1)	..	..	..	..	..	1
Diphtheria	6(3)	3(3)	3(2)	1(1)	3(3)	..	..	..	16
Dysentery (Amœbic)	*	1(1)	..	..	..	..	..	..	1
Dysentery (Bacillary)	*	..	2(1)	..	1(1)	..	..	..	3
Encephalitis Lethargica	..	..	..	..	1(1)	..	..	..	1
Erysipelas	*	*	..	..	*	*	..	..	..
Filariasis	*	..	..	..	..	..	..	..	..
Helminthiasis	*	..	*	*	*	*	*	*	..
Hydatid	*	..	*	*	..	*	*	..	..
Influenza	*	*	*	..	*	*	..	*	..
Lead Poisoning	*	*	..	*	*	*	*	*	..
Leprosy	..	..	..	..	..	..	..	..	..
Malaria(*b*)	*	..	2(2)	..	..	..	..	..	2
Measles	*	*	*	389(258)	*	*	..	..	389
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	18(2)	1	3	3(2)	..	..	..	..	25
Psittacosis	*	..	*	..	*	*	*	*	..
Puerperal Fever	..	*	..	..	..	..	..	..	..
Rubella(*c*)	*	..	..	*	1(1)	1(1)	..	..	2
Scarlet Fever	20(9)	32(15)	8(8)	5(2)	6	..	..	..	71
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	*	..	1(1)	*	*	*	*	*	1
Trachoma	*	..	*	*	*	*	*	*	..
Tuberculosis(*d*)	19(17)	27(22)	13(?)	8(6)	13(10)	3(1)	..	..	83
Typhoid Fever(*e*)	1	..	..	..	..	..	..	..	1
Typhus (Endemic)(*f*)	..	..	2	..	..	..	..	..	2
Undulant Fever	..	1(1)	..	..	..	..	..	..	1
Weil's Disease(*g*)	*	*	5	*	*	*	*	*	5
Whooping Cough	*	*	*	4(2)	*	*	..	..	4
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] The form of this table is taken from the *Official Year Book of the Commonwealth of Australia*, Number 37, 1946–1947. Figures in parentheses are those for the metropolitan area.

[2] Figures incomplete owing to absence of returns from the Northern Territory and Australian Capital Territory.

* Not notifiable.

(*a*) Includes Mossman and Sarina fevers. (*b*) Mainly relapses among servicemen infected overseas. (*c*) Notifiable disease in Queensland in females aged over fourteen years. (*d*) Includes all forms. (*e*) Includes enteric fever, paratyphoid fevers and other Salmonella infections. (*f*) Includes scrub, murine and tick typhus. (*g*) Includes leptospiroses, Weil's and para-Weil's disease.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Mellick, Richard Anthony, M.B., B.S., 1950 (Univ. Sydney), Lewisham Hospital, Lewisham.

Critoph, Ronald William, M.B., B.S., 1948 (Univ. Sydney), 1 Montague Road, North Cremorne.

The undermentioned have been elected as members of the New South Wales Branch of the British Medical Association:

Fields, Kim, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1945, District Hospital, Lithgow, New South Wales.

Flood, Martin Joseph, M.B., B.S., 1950 (Univ. Sydney), 42 Wilga Street, Concord West.

Fromer, Joseph, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1945, Immigration Centre, Greta, New South Wales.

Gillies, Douglas Neil, M.B., 1940 (Univ. Sydney), B.S., 1946 (Univ. Sydney), 26 William Street, Double Bay.

Hennessy, William Bertram, M.B., B.S., 1950 (Univ. Sydney), Saint Vincent's Hospital, Darlinghurst.

Jacobi, Marian, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1945, Mental Hospital, Orange, New South Wales.

Keating, Bruce Ivor, M.B., B.S., 1950 (Univ. Sydney), Lewisham General Hospital, Lewisham.

Kyneur, Frederick James, M.B., B.S., 1950 (Univ. Sydney), Saint Joseph's Hospital, Auburn.

Mackay, Elizabeth Kathleen, M.R.C.S. (England), L.R.C.P. (London), 1922, The L.M.S., Mission House, Port Moresby, Papua.

Niedzinski, Jozef, registered in accordance with the provisions of Section 17 (1) (c) of the *Medical Practitioners Act*, 1938-1945, 79 Ocean Street, Woollahra.

Pittorino, Anthony Joseph, M.B., B.S., 1950 (Univ. Sydney), Saint Joseph's Hospital, Auburn.

Schneeweiss, Joachim, M.B., B.S., 1950 (Univ. Sydney), Marrickville District Hospital, Marrickville.

Wilkinson, Thomas, M.B., 1950 (Univ. Sydney), Flat 2, 128 Johnston Street, Annandale.

Wilson, Lionel Leopold, M.B., 1950 (Univ. Sydney), Balmain and District Hospital, Balmain.

Chambers, Ella, M.B., B.Ch., 1927, Queen's University (Belfast), North Haven, Laurieton, N.S.W.

Corrigendum.

IN the paper by K. H. Heard, A. H. Campbell and J. J. Hurley on hypokalæmia complicating sodium para-aminosalicylate therapy for pulmonary tuberculosis published in the issue of October 21, 1950, an error appears on page 608. In the eighth line of the first column the figures 12·5 should read 21·5 (representing the patient's serum potassium content).

Medical Appointments.

Dr. Lawrence William Alderman has been appointed a Justice of the Peace for the Northern Territory.

Dr. G. H. Henry has been appointed Officer of Health for Iron Knob and District, South Australia.

Dr. L. T. Wedlick has been appointed a member of the Masseurs Registration Board of Victoria.

Dr. M. L. D. McKeon has been appointed Government Medical Officer at Bundaberg, Queensland.

Dr. R. J. Nash has been appointed Medical Officer, State Government Insurance Office, Brisbane.

Diary for the Month.

Nov. 1.—Western Australian Branch, B.M.A.: Council Meeting.
Nov. 1.—Victorian Branch, B.M.A.: Branch Meeting.
Nov. 2.—South Australian Branch, B.M.A.: Council Meeting.
Nov. 3.—Queensland Branch, B.M.A.: Branch Meeting.
Nov. 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 10.—Queensland Branch, B.M.A.: Council Meeting.
Nov. 13.—Victorian Branch, B.M.A.: Finance, House and Library Subcommittee.
Nov. 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 15.—Western Australian Branch, B.M.A.: General Meeting.
Nov. 16.—Victorian Branch, B.M.A.: Executive Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney)—All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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